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HYPERTHYROIDISM ASSOCIATED WITH HYPOPOTASSAEMIC PERIODIC PARALYSIS

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The association of hyperthyroidism with hypopotassaemic periodic paralysis is extremely rare. Since the first description nearly 60 years ago[1] only 40 cases have been described. In 1955, Linder[2] reviewed the literature and found 35 case reports to which he added 3 further cases. Subsequently Overholt *et al.*[3] and Anderssen and Wigmostad[4] have each described single cases. The object of this paper is to report another instance of this rare association. As far as we are aware it is also the first case to be associated with a positive thyroid auto-antibody test.

CASE REPORT

A 53-year-old European man, an advertising executive, was seen for the first time at 10 a.m. on 10 March 1958. Ten hours previously he had woken and found he was unable to sit up or move his limbs. During the previous 2½ years he had experienced frequent episodes of weakness and stiffness of his legs. In March 1956, 2 months after recovering from a severe bout of gastro-enteritis, he suddenly collapsed while walking and was so weak that he was unable to lift himself up. Similar attacks, which were transient, recurred 2 or 3 times daily for about 10 days and then disappeared spontaneously. During the next 2 years milder attacks of weakness and stiffness of the legs occurred. He was particularly liable to an attack if he sat in one position for more than 15 minutes, when he would find great difficulty in rising. Attacks could be prevented by changing his position at frequent intervals and were alleviated by exertion which 'loosened him up'.

Apart from these mild episodes he had woken on 3 occasions with attacks of weakness similar in distribution, but milder than the present episode. He had always recovered spontaneously by the following morning. The proximal muscles of the limbs were more severely affected and he had not experienced any ocular, bulbar, respiratory or sphincter disturbance. There were no particular precipitating factors, but the attacks tended to occur in the evenings, during weekends or when he was anxious. For the past 35 years he had passed frequent, loose stools on several days of each week. In 1932 a laparotomy had been performed because of this, and his appendix had been removed. He did not use purgatives and had never noticed any relation between exacerbations of his diarrhoea and the episodes of weakness or paralysis. During the past year he had become nervous and shaky, sweated excessively, and had lost 10 lb. in weight. The remainder of the history was not contributory.

Examination showed an anxious, sweaty, middle-aged man with warm extremities. The heart rate was 120 per minute and the blood pressure was 120/60 mm.Hg. Apart from a grade 1 apical systolic murmur there were no findings of note in the heart, chest or abdomen. The thyroid gland was slightly enlarged, smooth and firm. No bruits were heard over it. The cranial nerves and fundi were normal and there were no signs of bulbar or respiratory weakness. The patient was unable to sit up and there was symmetrical weakness of all limbs, most marked proximally. The hands and feet could be flexed and extended, but no other limb movements were possible. Muscle tone was moderately reduced and the tendon reflexes were absent. The affected muscles felt normal. There was no wasting, fasciculation or sensory loss. Rectal examination was normal.

Investigations at this time gave the following results: haemoglobin 16·2 G. per 100 ml.; haematocrit 50 vols. %; ESR (Wintrobe) 5 mm. in 1 hour; leucocytes 13,700 per c.mm. with a normal differential count; serum sodium 141 mEq. per litre; and potassium 2·7 mEq. per litre. Urine examination was normal.

No immediate treatment was given and within 3 hours spontaneous improvement began. Eight hours later the patient could walk with difficulty. At this time potassium chloride was given in doses of 15 gr. *t.d.s.* and was continued for 2 days. He was then well without any weakness. A tachycardia of 100 per minute and a fine tremor of the fingers were present.

One week after the onset, X-ray examination of the heart and lungs was normal, as was the electrocardiogram. Blood urea was 23 mg. per 100 ml.; plasma CO_2-combining power 24; serum sodium 144, potassium 4·9 and chlorides 108 mEq. per litre. Serum proteins were 7·8 G. per 100 ml., of which albumin was 3·5 and globulin 4·3 G. per 100 ml. Urine examination on several occasions showed random specific gravities between 1016 and 1020 and pH ranged between 4·9 and 5·5; no abnormal constituents were present. The serum protein-bound iodine (PBI) was 9·3 µg. per 100 ml.; the I^{131} uptake at 5 and at 24 hours was 100% and the conversion ratio was 79%. The salivary sodium/potassium ratio was 1:1. An attempt was made to provoke an attack of paralysis by the ingestion of 150 G. of glucose, but this was unsuccessful. Intravenous pyelogram, barium meal and barium enema were all normal. On a normal diet the faecal fat was 2·6 G. in 24 hours and the serum calcium and phosphorus were 9·8 and 2·4 mg. per 100 ml. respectively.

On 4 April 1958 treatment with methimazole, 10 mg. *t.d.s.*, and potassium chloride, 15 gr. *t.d.s.*, was begun. One month later the patient was clinically euthyroid and the potassium chloride was stopped. The dose of methimazole was reduced to 5 mg. *t.d.s.* and continued for a further 11 months. Within 2 weeks of starting treatment he felt well and had no recurrence of stiffness, weakness or paralysis. This improvement has been maintained for more than 2 years and all signs of hyperthyroidism have disappeared; the serum PBI in September 1958 was 3·4 µg. per 100 ml. In June 1958 he had a severe bout of diarrhoea lasting 4 days, but this did not cause any muscle weakness or hypopotassaemia. In 1960 it became possible to have tests for thyroid antibodies performed in Johannesburg. His serum showed a positive gel-agar precipitation test; the final titre was 1:8. The tanned red-cell agglutination test was negative.

DISCUSSION

Hypopotassaemia, regardless of aetiology, frequently causes muscle weakness or paralysis. This paralysis often has a typical distribution, affecting mainly the proximal muscles of the limbs. The affected muscles may swell and feel firm. The tendon reflexes are diminished or absent.[5] Chronic potassium depletion often causes renal dysfunction, characterized by polyuria, nocturia, hyposthenuria and inability to produce urine with a low pH. It is often associated with a metabolic alkalosis.[6]

Hypopotassaemia may result from excessive losses of potassium ions through the gastro-intestinal or urinary

tracts or from alterations in the distribution of potassium within the body. Although chronic diarrhoea may have contributed to the hypopotassaemia in this patient, his rapid spontaneous clinical recovery, the absence of renal impairment and the subsequent normal serum levels without potassium supplements, suggest that there was no depletion of the body's stores of this ion. His subsequent progress has shown complete relief of muscular symptoms despite an unchanged bowel habit. Primary hyperaldosteronism[7] was also considered unlikely, in view of the normal blood pressure and salivary electrolyte ratio and the absence of evidence of chronic potassium depletion. 'Potassium-losing nephritis' was excluded by the normal urinary sediment, pH and concentrating powers, and the normal blood urea and CO_2-combining power. The absence of chronic potassium depletion also made this diagnosis seem unlikely. It seemed probable that the hypopotassaemia was due to a shift of ions from the extra- to the intracellular phase.

Periodic paralysis is an uncommon condition manifested by recurrent episodes of muscle weakness and stiffness. Cardiac and respiratory involvement has occurred only rarely. The majority of cases are familial, but a few sporadic cases have been described. The sporadic and familial types differ in certain respects. The familial type usually begins at puberty and has a slight male preponderance. Treatment is usually unsatisfactory, but the condition tends to remit in middle-age.[5] The sporadic type usually begins after the age of 40 and is found almost exclusively in males. The prognosis of sporadic cases associated with thyrotoxicosis is good, since cure of both conditions is often achieved when the hyperthyroidism is controlled. Factors which provoke attacks are common to both the familial and sporadic types and include exertion, prolonged sitting, sleep, and meals rich in carbohydrates.

Most investigators believe that periodic paralysis is due to changes in the muscle cells, although the precise chemical abnormalities have never been proved. A transient fall in serum-potassium levels during paralytic episodes has often been detected, but this finding has not been invariable.[9] This reduction is probably due to a shift of potassium ions into the muscle cells. Szent-Gyorgyi[10] suggested that the increased potassium concentration in the muscle cells prevented the combination of actin and myosin necessary for muscle contraction. Recently Grob *et al.*[11] have shown that in periodic paralysis, skeletal muscle responds abnormally to the administration of glucose and insulin. The ratio between intra- and extracellular potassium ions changes, with a resultant increase in the resting muscle membrane potential and diminished contractility. Conn *et al.*[12] related the muscle changes to alterations in sodium metabolism as well. They suggested that during paralytic episodes temporary hyperaldosteronism occurs with an increase in the sum of intracellular sodium and potassium ions. In their patients a low sodium diet could prevent induced or spontaneous attacks.

Apart from the cases previously mentioned, Okinaka *et al.*[13] discussed the association of thyrotoxicosis and periodic paralysis in Japan. Their report was based on questionnaires sent to 6,333 patients who had undergone thyroidectomy operations between 1937 and 1956. Among 1,250 males, 110 (8·9 %) were thought to have suffered from periodic paralysis before their operations. Only 0·4% of the female patients were similarly affected. The retrospective nature of this inquiry and the lack of personal observation raise doubts whether all these patients really suffered from hypopotassaemic periodic paralysis. Only cases treated surgically were considered and the exclusion of cases treated by other means may have created a falsely high incidence of this association. Nevertheless, this association of conditions is possibly commoner in Japan than elsewhere.

Although periodic paralysis is an uncommon syndrome, we feel that any patient who develops the sporadic form, particularly in middle-age, should be investigated to exclude hyperthyroidism. Where the conditions are associated, the paralysis is usually cured by successful treatment of the thyroid disease.

SUMMARY

A case of hypopotassaemic periodic paralysis associated with thyrotoxicosis is described. This association is very rare. Treatment of the hyperthyroidism produced a gratifying cure of the disabling muscular symptoms.

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Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

VAN DIE REDAKSIE : EDITORIAL

DIE BEHANDELING EN HANTERING VAN PARAPLEGIESE PASIËNTE

Vir baie geneeshere en ander persone wat betrokke is by die behandeling en hantering van paraplegiese pasiënte, roep die woorde, ‚paraplegiese pasiënt' die beeld op van iemand wat aan die bed gekluister is, wat groot oop bedsere het, wat met 'n inblywende kateter aan 'n bottel sleg-ruikende urien gekoppel is, en wat onderhewig is aan pynlike spierkrampe. Tot nog maar onlangs was hierdie toestand van sake werklik van toepassing op pasiënte met ernstige siekte van die rugmurg, en tussenkomende infeksie het maar alte dikwels die genadeslag en verlossing van die lyding meegebring.

Uit die artikel van dr. McMurray[1] oor ‚The treatment and rehabilitation of traumatic paraplegic patients in South Africa' en die brief van dr. Jacobson[2] in antwoord daarop, wat vroeër vanjaar in die *Tydskrif* verskyn het, blyk dit hoe 'n groot behoefte daar nog in hierdie land bestaan aan spesiaal-toegeruste eenhede waar paraplegiese pasiënte behandel kan word. Elders bestaan daar wel sulke sentrums wat spesiaal uitgerus en met personeel toegerus is om probleme in hierdie verband te hanteer. Een van die meer bekende inrigtings van dié aard is die Stoke Mandeville-hospitaal by Aylesbury in die Verenigde Koninkryk. Guttman en sy kollegas[3] het daar baie voorbereidingswerk gedoen. Die hantering van paraplegiese pasiënte vereis die entoesiastiese samewerking van geneeshere, fisioterapeute, en verpleegsters op 'n groter skaal as in enige ander vertakking van die medisyne. Want dit is slegs deur spanwerk dat goeie resultate behaal kan word en dat die lewe van die pasiënt draagbaar gemaak kan word—dat selfs 'n mate van selfstandigheid deur die pasiënt bereik kan word.

Daar is baie aspekte aan die probleem van die paraplegiese pasiënt verbonde, bv. beheer van pyn, spierkramp en verkrimping, bedsere, blaasfunksie, en die laaste maar nie die minste nie, rehabilitasie wat betref die geestestoestand van die pasiënt. Platt en sy medewerkers[4] het die voorkoming van hierdie toestande beklemtoon, maar die aanvang van komplikasies is dikwels stadig, en as hulle eers gevestig is, ontwikkel hulle vinnig sodat die pasiënte by die hospitaal beland met ernstige bedsere en growwe misvormings.

Tans word twee metodes gebruik om spastisiteit en sy meegaande euwels te verlig. Eerstens kan genoem word 'n gekombineerde operasie wat bestaan uit obturator-neurektomie vir die verligting van adduktor-spasma, oorplanting van die spiertendon van die haksenings na die knobbels van die femur (om buiging van die knie in strekking te verander[5]), en eenvoudige tenotomie van die achilles-tendon om spastiese voetsoolbuiging te verlig. Platt *et al.*[4] het goeie resultate met hierdie metode bereik. Afgesien van die voor-die-hand-liggende nadele van chirurgiese prosedures by hierdie soort pasiënt, gee dié operasie ook aanleiding tot 'n neurologiese tekort, en in baie gevalle verander dit net eenvoudig 'n spastiese ledemaat in een wat slap is.

Meer onlangs is die gebruik van intratekale fenol in 'n medium soos gliserien of ‚miodil' beskryf. Die idee om 'n chemiese toksien vir 'n perifere senuwee te gebruik, is nie nuut nie, en sulke stowwe soos alkohol is in die verlede gebruik. Fenol het baie voordele bo ander toksines wat vinniger werk. Met ondervinding kan dit gelokaliseer word tot 'n paar anterior of posterior wortels, en daarbenewens is sy aksie nie onmiddellik en onomkeerbaar nie. Dit skyn ook 'n selektiewe uitwerking te hê op die geleiding van pyn, sonder om ander sensoriese modaliteite aan te tas.[6,7] Motoriese krag word soms verbeter deur die verligting van sterk spastisiteit, iets wat die oorblyfsels van willekeurige krag kan bedek. Daar is egter een groot nadeel by die gebruik van fenol, waarop alle skrywers wys, naamlik, inmenging met blaasfunksie. Dit gebeur as die fenol die rugmurg self bereik of afsak tot by die sakrale wortels. Met die voorbehoud dat dié gebeurlikheid verhoed kan word, kan die gebruik van fenol werklik tot voordeel wees by die verligting van pyn.

Daar is nie algemene ooreenstemming oor die beste metodes om bedsere te behandel nie. Doeltreffende verpleging is waarskynlik die belangrikste faktor. Dit is treffend om te sien hoe 'n diep, aktiewe seer kan opklaar met geduld en toewyding.

Die hantering van blaasfunksie is alreeds uitvoerig bestudeer deur neurofisioloë, neuroloë, en uroloë. Geen twee gevalle van rugmurgsiekte is egter eenders nie. Infeksie van die urienweë moet, indien moontlik, voorkom word, maar dit is selfs onder die beste omstandighede nie altyd moontlik nie. Veel kan egter tog gedoen word om die lewe van die pasiënt draagbaar te maak. Die doel moet wees om 'n outomatiese blaas te verkry in gevalle van hoë algehele afsluiting van die rugmurg. In ander gevalle mag daar oorblyfsels van willekeurige beheer wees, en alle pogings moet in die werk gestel word om dié funksie te probeer bewaar. 'n Klein spastiese blasie moet vermy word, en pasiënte moet aangemoedig word om urien in toenemende hoeveelhede te behou om die ‚muur' van die blaas te rek.

Saam met die rehabilitasie van liggaamlike funksies is daar die geestesaanpassing van die pasiënt wat in gedagte gehou moet word. Dit is 'n besondere belangrike faset van die onderhawige probleem. Baie pasiënte slaag wel daarin om hul groot gebrek te aanvaar—'n toestand van sake wat veral geld in gevalle van traumatiese skade aan die rugmurg. Dit is slegs nodig om 'n eenheid vir die behandeling van rugmurg-toestande te besoek om die hoë vlak van moed en volharding van die pasiënte en hul versorgers te sien en om te besef watter bevrediging daar spruit uit die doeltreffende en suksesvolle hantering van hierdie probleem.

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THE MANAGEMENT OF PARAPLEGIC PATIENTS

To most medical attendants the word 'paraplegia' conjures up the image of a bedridden patient beset with gaping bedsores, connected by an indwelling catheter to a bottle of foul urine, and racked by painful muscle spasms. Until fairly recently this indeed was the fate of those patients with severe spinal cord disease. Inevitably death from an intercurrent infection provided a merciful release from great suffering.

Mr. McMurray's article[1] on 'The treatment and rehabilitation of traumatic paraplegic patients in South Africa' and Mr. Jacobson's letter[2] in reply to some of the views expressed by Mr. McMurray, which were published in this *Journal* some time ago, served as urgent reminders of the dire need for paraplegic centres in this country. Elsewhere there are centres expressly equipped and staffed to deal with the considerable problems of management. One of the better known of these centres is that which is at Stoke Mandeville Hospital near Aylesbury in the United Kingdom. Much pioneer work has been done there by Guttman[3] and his colleagues. The management of paraplegic patients requires the enthusiastic cooperation of doctors, physiotherapists, and nurses to a greater extent than in any other branch of medicine. For it is by teamwork alone that good results can be achieved and the patient's life made bearable or even restored to some measure of independence.

There are many aspects to the problem of the paraplegic patient, eg. control of pain, muscle spasm and contracture, bedsores, bladder function, and last, but by no means least, the mental rehabilitation of the patient. The prevention of these states has been stressed,[4] but unfortunately the onset of complications is often insidious, and once established these proceed with remarkable rapidity so that patients arrive at hospital with gross bedsores and severe deformities.

Two methods for the relief of spasticity and its concurrent evils have been in recent use. Firstly, a combination operation consisting of obturator neurectomy for the relief of adductor spasm, transplants of the hamstring muscle tendons to the femoral condyles to convert knee flexion to extension,[5] and simple tenotomy of the Achilles tendon to relieve spastic equinus, has been used. Good results from this method of treatment has been recorded by Platt *et al.*[4] However, apart from the obvious disadvantages of any surgical procedure in these patients, this method does add to the neurological deficit and, in many instances, simply converts a paralysed spastic limb to a paralysed flaccid one.

More recently the use of intrathecal phenol in a vehicle such as glycerol or 'myodil' has been described. The idea of using a chemical toxin on the peripheral nerve is not new and in the past substances such as alcohol have been used. Phenol has many advantages over other more rapidly acting toxins. With experience it can be localized to only a few anterior or posterior roots and, moreover, its action is not immediate and irreversible. In addition it appears to have a selective action on pain transmission without noticeably affecting other sensory modalities.[6,7] Motor power is sometimes improved by the relief of extreme spasticity which may mask any remains of voluntary power. There is however one major drawback to the use of phenol which is noted by all authors, and that is interference with bladder function. This occurs when phenol reaches the cord itself or descends to the sacral roots. Provided this can be avoided, the use of phenol for relief of pain and spasticity appears to have real advantage.

There is no firm agreement on the best method for treating bedsores. Efficient nursing plays the major rôle. It is remarkable how a deep, fungating ulcer can be made to heal with care and patience; and this provides visible evidence of devoted nursing.

The management of bladder function has been extensively studied by neurophysiologists, neurologists, and urologists. Suffice it to say that in spinal cord disease no two cases are alike. If possible, urinary infection is to be avoided, but unfortunately this is unlikely even in the best of hands. Yet much can be done to make the patients' life more tolerable. The aim is to produce an automatic bladder in those cases where there is complete, high cord disease. In others there may be remnants of voluntary control and every effort should be directed towards preserving this function. A small spastic bladder is to be avoided, and patients should be encouraged to try to retain urine in increasing amounts in order to stretch the bladder wall.

Simultaneously with the rehabilitation of bodily function, the adjustment of the patients' mental outlook must be considered. This is an extremely important facet of the problem under consideration. The majority of patients manage to come to terms with their great disability. This is especially so in cases of traumatic spinal cord damage. One has only to visit a spinal cord unit to witness the high morale of these patients and their attendants to realize the singular gratification provided for all concerned by the successful management of this problem.

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THE SOUTH AFRICAN MEDICAL CONGRESS, 24 - 30 SEPTEMBER 1961, CAPE TOWN

HOTEL ACCOMMODATION

The Forty-third Medical Congress of the Medical Association of South Africa will be held in *Cape Town* from 24 to 30 September 1961. Information regarding the Congress is published at regular intervals in the *Journal*. Although it is still relatively early, members who intend coming to Congress are requested to send in their Intention Forms as soon as possible.

Prospective visitors to the Congress are also requested to bear in mind that *hotel accommodation in Cape Town is somewhat limited* and that they should make the necessary reservations well in advance of Congress. The Travel Bureau of the South African Railways has been appointed the official agent in this connection. Members are requested to contact their local agents who have been extensively circularized in this respect. In the small platteland towns the nearest stationmaster will handle the matter.

Make your reservations now in order to avoid disappointment. In the event of any difficulty, please write to Dr. J. C. Coetzee, Convener, Accommodation Sub-Committee, 43rd South African Medical Congress, P.O. Box 643, Cape Town.

AORTO-ILIAC OCCLUSIVE DISEASE*

A REPORT ON 67 CONSECUTIVE CASES TREATED SURGICALLY

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(*Continued from page 375 of the* Journal *for 6 May, 1961*)

TREATMENT

We do not propose discussing treatment in detail, but wish to mention the types of operation performed and the results obtained. We cannot emphasize too strongly that surgical treatment is only part of the overall management of these patients, who require life-long observation and careful supervision. In all of them careful instructions must be given in regard to abstinence from smoking, foot care, low-lipid diet and graduated exercises. Those with involvement of the distal vessels may have to continue with Buerger's exercises and the general cardiovascular state may require special attention. It is our practice to commence anticoagulant therapy 24 - 27 hours after operation and to continue with it indefinitely unless there are contra-indications. We do not prescribe antispasmodics in patients with occlusive vascular disease.

OPERATIONS

The types of operation performed included thrombo-endarterectomy (56), thrombo-endarterectomy combined with excision and grafting (6), bypass grafting (3), and sympathectomy (2) — see Table VII.

TABLE VII. TYPE OF OPERATION PERFORMED IN RELATION TO NATURE OF LESION

	A	*AI*	*I*	*IF*	*AIF*	*Total*
Number of cases	7	15	6	6	33	67
Endarterectomy	6	13	6	5	26	56
Endarterectomy and graft	—	2	—	—	4	6
Graft	1	—	—	1	1	3
Sympathectomy	—	—	—	—	2	2

A, AI, I, IF, AIF as in Table III.

Our bias has been towards thrombo-endarterectomy, and simple bypass grafting was rarely performed. The latter procedure was carried out in our first case with a localized aortic bifurcation block and in 2 patients who had additional involvement of the superficial femoral artery. In 1 of these 2 patients the graft was taken from the common iliac to the popliteal with side-to-side anastomosis to the common femoral. All the grafts were of woven, crimped 'teflon'.

In 6 patients thrombo-endarterectomy of the proximal vessels was combined with distal ilio-femoral or ilio-femoro-popliteal grafting. In 5 of them this was necessary because of damage to the iliac vessels by the stripper, and in 1 the graft was used to bypass an occluded superficial femoral artery.

'Extended lumbar sympathectomy' with removal of the ganglia from T9 to L3 inclusive was done in 2 patients. Both had extensive disease with calcification and were considered technically unsuitable for direct arterial surgery.

* Paper presented at the Annual Congress of the South African Orthopaedic Association (M.A.S.A.), Cape Town, 13 October 1960.

RESULTS

Early Results (Table VIII)

The results were classified as excellent or improved only when there was both objective and subjective clinical improvement as well as a significant improvement in oscillometry (Fig. 21) and/or plethysmography (Fig. 22).

TABLE VIII. RESULTS OF SURGERY—EARLY

	A	*AI*	*I*	*IF*	*AIF*	*Total*	
Number of cases	7	15	6	6	33	67	
Local:							
Excellent	6	8	5	2	11	32	56 (83%)
Improved	1	7	1	2	13	24	
Unchanged	—	—	—	1	4	5	11
Unchanged but amputation	—	—	—	1	2	3	
Worse	—	—	—	—	1	1	
Worse and amputation	—	—	—	—	2*	2	
Pulses:							
Returned	4	6	3	4	5	22	45 (67%)
Improved	2	5	1	—	5	13	
Unchanged present	1	4	2	1	2	10	
Unchanged absent	—	—	—	1	19	20	
Disappeared	—	—	—	—	2	2	

* One of these patients died subsequently.
A, AI, I, IF, AIF as in Table III.

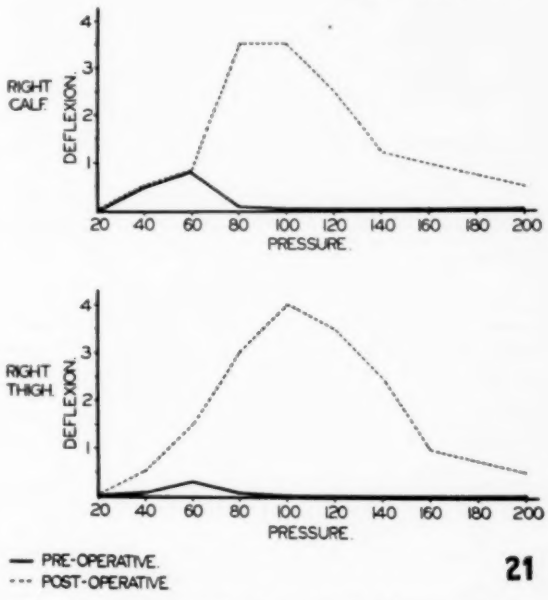

Fig. 21. Oscillometry showing a marked improvement in pulsation in right calf and thigh following the removal of an occlusion at the aortic bifurcation.

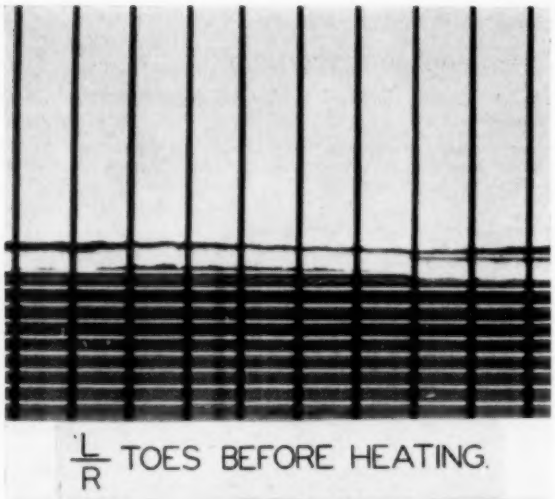

Fig. 22. Plethysmography demonstrating significant improvement following aorto-iliac endarterectomy. Note: Upper graph — before operation; lower graph — after operation.

On this basis the results in 56 (83%) of the patients were regarded as successful. It should be noted that successful results were obtained in every one of the patients with localized aortic, aorto-iliac or iliac blocks.

Postoperatively, pedal pulses were palpable in 45 patients but were classified as 'returned' or 'improved' in 35 only. This is not surprising because the femoral vessels were involved in 39 (60%) of our patients. Indeed, pedal pulses could be felt in all of the 28 patients who had localized blocks, although they were regarded as 'returned' or 'improved' in 21 only.

The results were unsuccessful in 11 patients and all of them had associated femoral occlusions. In 8 the condition of the limb remained unchanged; 3 of them required amputations 1 - 6 weeks later, but this would have been necessary anyway. In 3 patients surgery aggravated the ischaemia. One of them required amputation after a week and another developed extreme ischaemia of both limbs necessitating amputation of 1 limb after 8 days. This patient died 20 days after the original operation.

Late Results (Table IX)

Our patients have not been followed-up for a sufficiently long period to justify any claims in regard to success. Nevertheless, there are some points of interest.

The follow-up period ranged from 4 to 22 months with an average of 7½ months. In 31 the period was less than 6 months; in 20 from 6 to 12 months; and in 15 from 12 to 24 months.

TABLE IX. RESULTS OF SURGERY—LATE

Number of patients	A 7	AI 15	I 6	IF 6	AIF 33	Total 67
Early successes:	7	15	6	4	24	56
Relapse	—	4	—	—	5	9
Re-operation	—	2	—	—	3	5
Late result:						
Success	7	12	6	3	23	51
Deteriorating	—	2	—	—	1	3
Amputation	—	—	—	1	—	1
Death	—	1	—	—	—	1
Early failures:	—	—	—	2	9	11
Early death	—	—	—	—	1	1
Re-operation	—	—	—	—	4	4
Late result:						
Success	—	—	—	—	2	2
Unchanged	—	—	—	1	4	5
Deteriorated	—	—	—	—	2	2
Death	—	—	—	1	—	1

A, AI, I, IF, AIF as in Table III.

Second operations were done in 9 patients — in 5 for a relapse after initial success and in 4 for progressive deterioration after initial failure. Two had localized aorto-iliac occlusions and 7 had extensive aorto-ilio-femoral disease. One of the patients died of renal failure and 2 were not improved, but the remaining 6 are much improved and symptom-free.

Of the 56 patients in whom early successes were obtained, the subsequent progress was as follows:

1. Forty-seven remained symptom-free with good circulation in both limbs.

2. Five had major relapses due to re-thrombosis of the iliac vessels. Two of these had localized aorto-iliac occlusions and 3 had diffuse aorto-ilio-femoral disease. All were operated upon for a second time with success in 4. There was 1 postoperative death due to a renal 'shut-down'.

3. Three have gradually deteriorated with disappearance of pulses which had been present. Two of them had localized aorto-iliac thrombosis and one had aorto-ilio-femoral occlusion.

4. One developed a secondary haemorrhage from the femoral anastomosis of an ilio-femoro-popliteal bypass graft for ilio-femoral occlusion 2 months after the operation and required an amputation 1 month later.

Thus, of the original 56 successes, 51 are now symptom-free, 3 have deteriorated, one has required an amputation and one has died after a re-operation.

Of the 11 patients in whom the early results were unsuccessful, 1 died early and the subsequent progress in the remaining 10 was as follows:

1. Four were re-operated upon because of progressive deterioration. All of them had aorto-ilio-femoral occlu-

sions. Two improved after re-operation but the other 2 continued to deteriorate.

2. One who had had an early amputation for ilio-femoral occlusion, died of a coronary thrombosis 2 months later.

3. Five have remained unchanged. Three of these had had amputations for aorto-ilio-femoral occlusions. One had diffuse aorto-ilio-femoral disease treated by sympathectomy and 1 had ilio-femoral occlusion.

Thus of the 10 early failures who have been followed-up, 2 have been improved, 5 are still unchanged, 2 have deteriorated despite re-operation and 1 has died of coronary thrombosis.

Overall Results (Table X)

The overall results, summarized in Table X, show that our initial success rate was 83% and the later success rate 79%. The initial mortality was 1·5%, but since then it has increased to 4·5%. Early amputations were required in 6% and this figure has increased to 7·5%. No doubt a longer follow-up will reveal even more late failures although many of them may still benefit from second or even third operations. However, the first 15 patients in this series have now been followed up for more than a year and the results are still successful in 12 (80%). The 3 failures all had diffuse aorto-ilio-femoral disease. One of them required an early amputation and the initial results in the other 2 were also unsuccessful. Our results, therefore, compare favourably with those of others.[10,15,23,25,26] In general, the success rate of operations on aorto-iliac occlusions after 18 - 24 months appears to be 70 - 80% and the initial mortality 2 - 5%.

TABLE X. RESULTS OF SURGERY—SUMMARY

	Without femoral involvement	*With femoral involvement*	*Total*
Number of patients	28	39	67
Early results:			
Successful	28 (100%)	28 (72%)	56 (83%)
Amputation	—	4	4
Death	—	1	1
Late results:			
Successful	25 (89%)	28 (72%)	53 (79%)
Amputation	—	1	1
Death	1	1	2
Total amputations	—	5	5
Total deaths	1	2	3

It should be noted that, in patients with localized segmental occlusions (aortic, iliac or aorto-iliac), the initial success rate was 100% with no mortality and the later success rate 89% with 1 (3·6%) death and no amputations. These figures approximate the phenomenal successes claimed by Crawford *et al.*[10] and contrast strikingly with the results in patients suffering from diffuse occlusions. Femoral occlusion is probably the most important single factor to lower the success rate. Firstly, it compromises an adequate 'run off' and so predisposes to early thrombosis. Secondly, about 50% of the grafts will close later.[25,26]

Extensive disease of the external iliac has also been an adverse factor, in our experience, mainly because it is usually accompanied by femoral occlusion. Thus in 6 patients with ilio-femoral occlusions initial successes were obtained in 4, while 1 remained unchanged and 1 required an amputation. Later, the amputee died and 1 of the early successes relapsed and required an amputation. In the same way extensive involvement of the external iliac in patients with diffuse aorto-ilio-femoral occlusions compromised the results.

Distal-vessel thrombosis undoubtedly affected the result adversely, but was not always easy to detect either clinically or radiographically. However, in general the patients who had foot claudication, severe rest pain and major ischaemic lesions, did not do as well as the others.

DISCUSSION

From the above it should be clear that patients with localized segmental blocks are more suitable for direct arterial surgery than those with diffuse disease. Not only are the results of surgery considerably better in the group with localized disease, but the incidence of associated cardiovascular disease and hypertension is less; consequently they are more favourable surgical risks.

The vital question, however, is whether surgery is indicated or indeed justified for segmental occlusions. It must be remembered that, until medical or dietary measures can control the inevitable progression of atherosclerosis, this type of surgery is no more than palliative.[12] Many patients, treated conservatively, continue to lead useful lives for long periods with no more than mild, non-progressive symptoms.[12,27,29] De Wolfe *et al.*[13] followed 24 patients on conservative treatment for periods of 1 - 5 years and 11 improved, 10 remained unchanged and only 3 became worse. Singer and Rob[27] found that, of 22 patients treated conservatively over a period of 36 months, 20 were improved or remained unchanged. The long histories in many of our cases support this observation and so do Michael Boyd's[6] figures for the risk of amputation. In 1,440 patients who presented with claudication Boyd[6] found that the chance of amputation was 7·2% in the first 5 years and 12·2% in the first 10 years. He also found that none of those who had died succumbed from the effects of the local arterial lesion. Ill-advised surgery, which may end in the premature loss of a single life or even a single limb would, therefore, indeed be a tragedy.

On the other hand, there are some arguments in favour of early surgery. In the first place, many of the patients are comparatively young, and the restricted activity imposed upon them by this claudication may seriously affect their livelihood. Even Michael Boyd,[8] that great advocate of lumbar sympathectomy, admits that sympathectomy is of real value only in early grade 2 claudication. That would exclude 53 (78%) and probably 64 (95%) of the patients in our series and all those with localized blocks. Many of them found that their claudication was interfering with their work and direct arterial surgery was thus deemed justifiable.

In the second place there is the chance of further thrombosis. Although it is true that these patients do not progress rapidly from segmental to diffuse obstruction, which may be beyond the help of surgery,[12] Boyd[6] has found that the risk of further thrombosis is 20·9% in the first 5 years and 36·4% in the first 10 years; this will undoubtedly lessen the chances of successful surgery. Therefore, if there are any signs of progress, removal of a segmental occlusion seems fully justified.

Thirdly, there is the risk, only recently recognized and still unknown, of renal involvement with the development

of malignant hypertension. Our study of this aspect has been incomplete, but Starer and Sutton,[28] in a study of 32 consecutive cases of aortic thrombosis examined by aortogram, found the renal artery to be involved in 2. Fourthly, there is another unknown risk, namely, that of peripheral embolization from the aorto-iliac clot. These risks may yet prove to be sufficiently significant to justify early surgery, but at present we agree with Singer and Rob[27] that 'in many patients intermittent claudication is a relatively benign condition and that with proper selection, only a proportion of patients will require active surgery'.

In patients with diffuse aorto-iliac disease, particularly if associated with femoral involvement, the problem is different. Not only may we expect poorer results, but there is a significant incidence of associated coronary heart disease and hypertension. The risks of direct arterial surgery are, therefore, considerable, and many of the survivors are likely to succumb from cardiac, cerebral or interrecurrent disease within 5 - 10 years following operation. Michael Boyd[5] found that the chance of survival for 5 years was 73%, but that this chance was almost halved for 10 years (38·8%). 'Few of us would put a new engine into a car whose body was unlikely to see the year out; and likewise we should perhaps hesitate before undertaking grafting operations.'[2] Relieving the patient of his claudication is useless if his angina will not allow him to walk much further[6] or if his arthritis and emphysema keep him at home.

On the other hand, once secondary femoral thrombosis has developed, the ischaemia is very likely to progress to gangrene,[6] the claudication distance diminishes progressively to the point where the patient can hardly move and persistent rest pain interferes with his sleep. These are all ominous signs and, unless relieved by timely surgery before the popliteal and distal vessels have thrombosed, there is a grave risk that the patient will lose one or more limbs or simply fade away because of all the misery.[27] There can be no question that surgery is justified in these cases but the real problem is what type of surgery should be undertaken.

In Table XI our unsuccessful results in relation to the type of operative procedure are analysed. Comparisons

TABLE XI. UNSUCCESSFUL RESULTS IN RELATION TO TYPE OF OPERATION PERFORMED

	Endarterectomy	*Endarterectomy and graft*	*Graft*	*Sympathectomy*	*Total*
Number of patients ..	56	6	3	2	67
Early failures:					
Deaths	—	1	—	—	1
Amputations ..	3 } 4	1 } 4	— } 2	— } 1	4 } 11
Others	1	2	2	1	6
Later failures:					
Closure — successful re-operation ..	4	—	—	—	4
Closure—death ..	1	—	—	—	1
Haemorrhage — amputation	— } 7	— } —	1 } 1	— } 1	1 } 9
Relapse — distal closure	2	—	—	1	3

are not valid because of the small numbers treated by grafting and sympathectomy. Nevertheless, there are some observations which may be of interest.

1. *Endarterectomy* was attempted in 62 of the patients. It was technically possible to disobliterate the vessels in 56, with success in 52 (91%) and no early deaths. In 4 the vessels re-thrombosed early. Three of these patients lost their limbs — 2 in spite of surgery and 1 because of surgery. All the failures had diffuse disease with femoral involvement and a poor 'run off'.

In 5 patients the vessels re-thrombosed at a later date — all within 6 months. Two had localized aorto-iliac occlusions and 3 diffuse aorto-ilio-femoral occlusions. Two others with aorto-iliac occlusions developed later distal-vessel thrombosis.

The initial success rate was 91%, the late success rate 80%, and the overall surgical mortality 1·8%. This compares favourably with the results of others whose figures are 60 - 70% (initial successes), 60 - 70% (late successes), and 2 - 3% (mortality).[5] It should be noted that 7 of the 11 patients had femoral involvement with a poor 'run off'. It might therefore be argued that the results would have been improved by simultaneous femoral endarterectomy or bypass. This will be discussed below.

2. In 6 patients endarterectomy had to be supplemented by *grafting*. All these patients had diffuse disease with involvement of the external iliac and femoral vessels. In 5 our attempts at complete disobliteration damaged the vessels, which had to be replaced by grafts. In the sixth, endarterectomy was combined with an ilio-femoro-popliteal graft to bypass an occluded superficial femoral artery.

There were 4 early failures. One patient lost his life and another a limb because of surgery. There have been no later failures as yet, but these few cases seem to indicate that grafting after failed endarterectomy will not succeed. We have found that extensive endarterectomy from aorta to femorals and beyond is often extremely difficult, especially when the external iliac and femoral vessels are involved. This experience has convinced us that endarterectomy should rather not be attempted in these circumstances because failure carries the grave risk of loss of a limb or even of the patient's life. We certainly do not recommend extensive endarterectomy from aorta to popliteal for diffuse aorto-ilio-femoral occlusions. We also feel that endarterectomy of the external iliac should at all times be approached with caution and that it should be abandoned if any difficulty is encountered, lest the vessel be damaged and patent collaterals sacrificed. A method of treating diffuse disease that suggests itself is endarterectomy of aorta and common iliacs combined with ilio-femoro-popliteal or femoro-popliteal bypass grafting. Our experience of grafting after failed endarterectomy, however, makes us hesitate to follow this procedure and it would appear to be wiser to rely on primary bypass grafting or even sympathectomy.

3. Only 3 primary *bypass grafts* were done. All failed because of inadequate 'run off'. However, the ischaemia was not aggravated in any of the patients. We have been very satisfied with femoro-popliteal bypass grafts for femoral occlusions. Others claim excellent results for bypass grafting in aorto-iliac disease, the average figures being: mortality, 2 - 5%; initial success, 95%, and late success, 80%.[5] It would appear, therefore, that the insertion of grafts merits a trial particularly in diffuse disease when endarterectomy is not feasible. Unfortunately the initial success rate of bypass grafting is not maintained and may fall to 50%, due largely to the progress of the disease which interferes with the 'run off'.[25,28] Nevertheless, the operation has the great advantage that, if it fails, the

patient is none the worse; also the graft can be taken from the aorta to the popliteal artery if necessary. A most important factor for successful grafting, however, is a free distal outflow, and grafting, therefore, should not be done if the distal vessels are unsatisfactory. It should be noted that an adequate 'inflow' is as important as the outflow, and therefore simple femoro-popliteal grafting will not suffice in patients who have proximal disease as well as femoral occlusion.

4. *Lumbar sympathectomy* was done in 2 patients only, but in the past we have treated many patients by this method. One patient improved temporarily and the other remained unchanged. This is in keeping with our general experience with this method of treating diffuse atherosclerosis. However, we have recently done extended lumbo-dorsal sympathectomies (T9 - L3) with more promising results. This may yet prove to be the operation of choice in poor-risk patients and in those with extensive occlusive disease who are unsuitable for bypass grafting because of occluded distal vessels. We are now using it more frequently because 'there is a great deal to be said for being content to allow these patients to grow old gracefully'.[8] In certain patients the use of sympathectomy in addition to direct arterial surgery may improve the likelihood of continued function of the re-established circulation.[15]

In summary, therefore, we tentatively recommend the following:

1. Localized aortic, common iliac or aorto-iliac occlusions — thrombo-endarterectomy (only if the symptoms are incapacitating).

2. Localized external iliac occlusions — attempt endarterectomy but abandon if any difficulty occurs, and insert an ilio-femoral bypass graft (preferably from the end of the common iliac).

3. Ilio-femoral occlusions — bypass graft from aorta or common iliac to the popliteal with side-to-side anastomosis to the common femoral, provided the 'run off' at the popliteal is adequate. If the 'run off' is poor, perform lumbar sympathectomy.

4. Aorto-ilio-femoral occlusions —

(*a*) If the proximal disease is mainly in the aorta and common iliacs with a good backflow into the distal common iliac, and the popliteal 'run off' is good, combine proximal endarterectomy with simultaneous or later femoro-popliteal grafting or endarterectomy.

(*b*) If the proximal disease is more diffuse with external iliac involvement but the popliteal 'run off' is still good, insert an aorto-femoro-popliteal graft.

(*c*) If the patient is a poor surgical risk, or the disease is extremely diffuse with calcification, or the popliteal 'run off' is poor, perform bilateral 'extended' lumbar sympathectomy.

CONCLUSION

In recent years the scope of and indications for direct arterial surgery have continued to widen. Some surgeons are even bypassing and disobliterating normal arteries; this, no doubt, improves their overall results! Others regard vascular surgery as 'an initiation test to surgical manhood'[2] and dabble in disobliterating or joining arteries even before they have mastered the art of stripping veins or of intestinal anastomosis. Caution is obviously called for in the selection not only of patients but also of the surgeon.

In introducing this subject we made a plea that arterial thrombosis should always be considered in the differential diagnosis of every case of 'lumbago and sciatica'. In conclusion, our request is not to over-react by rushing into a diagnosis of vascular insufficiency without careful appraisal for, beware, 'the winds of change are blowing' and 'blocked arteries' rather than 'slipped discs' may become the vogue among backache sufferers. The onset of arteriosclerosis nearly always marks the beginning of a generalized degeneration in all tissues. Joint cartilages are wearing thin; spinal discs are degenerating; muscles are wasting and fibrositic conditions are widespread.[5] When these painful and disabling afflictions involve the lower back and limbs, a considerable problem is presented to both patient and doctor.

Before embarking on formidable surgical procedures, let us pay heed to the warning given by Lord Cohen of Birkenhead[6] who said: 'Backache is a symptom, not a disease, even when labelled 'lumbago' . . . It is a graveyard in which are buried once-fashionable methods of treatment which were founded on speculative pathology; the ghosts of these are restless and one must beware of their reappearance even when clothed in different garb'.

SUMMARY

The clinical features of 67 consecutive cases of aorto-iliac occlusive disease are described. Particular emphasis is laid on the many pitfalls which may be encountered in diagnosis, both clinically and by special investigation. The methods of treatment used in these cases are outlined and the results are analysed.

Indications for surgery in the localized and diffuse types of disease are discussed in relation to the results, and a scheme is presented outlining the type of operation recommended under particular circumstances.

The extreme care required for accurate diagnosis of aorto-iliac occlusive disease and for selection of patients for surgery is again emphasized.

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AN EPIDEMIC OF 'ACUTE EOSINOPHILIC PNEUMONIA' FOLLOWING 'BEER DRINKING' AND PROBABLY DUE TO INFESTATION WITH *ASCARIS LUMBRICOIDES*

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Patz[1] has recently described an epidemic of 'acute eosinophilic pneumonia' which occurred during November 1956 in 12 adult Bantu males at Middelburg, Transvaal. All the patients had attended a beer drink, illicitly held at a nearby brickfield, 6 days before the onset of symptoms. The illness started with a feeling of malaise, headache and generalized body pains, soon followed by rigors, pyrexia, and a severe cough productive of scanty white sputum. The patients were extremely breathless, and wheezing was a prominent feature. Rhonchi and fine crepitations were audible throughout the chest. From the 12th to the 17th day after drinking the beer the patients underwent the worst of their illness, but they then improved rapidly and were able to leave hospital after a further 5 days.

At least 3 patients had had a generalized urticarial rash

TABLE I. RELEVANT FEATURES OF THE 13 PATIENTS

Case and sex	*Date of drinking*	*Onset of respiratory symptoms*	*Date and place of examination*	*Stool for ascaris*	*Urticaria*	*Pyrexia*	*Leucocyte count*		*Eosinophils*		*Chest X-ray*
							Date	*c.mm.*	*%*	*Wet count*	
1 M	15.1.59	20.1.59	25.1.59 N.E.H. (admitted)	Negative 17.2.59. Negative 15.5.59. Negative 29.5.59	Present	Present	26.1.59 18.2.59	29,000 6,700	10 36	 2,200	Extensive bilateral mottling. Right interlobar effusion
2 M	15.1.59	20.1.59	24.1.59 C.H. admitted)	Positive 11.2.59	Present	Present	27.1.59 9.2.59	39,900 20,400	44 66		Extensive bilateral mottling. Interlobar effusions. Probable Kerley 'B' lines
3 F	15.1.59	22.1.59	25.1.59 C.H. (admitted)	Negative 10.5.59. Positive 29.5.59	Present	Present	26.1.59 18.2.59	16,400 6,300	4 12	 770	Extensive bilateral mottling. Interlobar effusion
4 M	18.1.59	25.1.59	29.1.59 N.E.H. (admitted)	Positive 18.2.59	Present	Present	30.1.59 16.2.59	9,600 6,100	3 21	 1,023	Increased hilar shadows. Right interlobar effusions. Mild mottling
5 F	15.1.59	20.1.59	30.1.59 (Ferndale)	Positive 18.2.59	Present	Not known	18.2.59	11,800	21	2,200	Not done
6 M	15.1.59	20.1.59	28.1.59 N.E.H.	Positive 18.2.59	Present	Present	29.1.59 18.2.59	19,500 4,400	36 12	 407	Mild bilateral mottling
7 M	18.1.59	25.1.59	1.2.59 B.H.	Positive 29.5.59	Present	Not known	21.2.59	8,900	7	638	Increased hilar shadows. Mild bilateral mottling
8 M	18.1.59	25.1.59	1.2.59 B.H.	Positive 18.2.59	Present	Not known	21.2.59	6,500	15	1,166	Increased hilar shadows. Interlobar effusions. Probable Kerley 'B' lines
9 F	15.1.59	24.1.59	31.1.59 N.E.H.	Negative 10.5.59. Positive 29.5.59	Present	Present	Not done				Normal (31.1.59)
10 F	18.1.59	21.1.59	1.2.59 C.H.	Positive 18.2.59	Present	Not known	21.2.59	6,500	15	990	Not done
11 M	15.1.59	Not known	5.3.59 (Ferndale)	Negative 17.2.59. Negative 15.5.59. Negative 29.5.59	Present	Not known	Not done				Not done
12 F	15.1.59	19.1.59	Not seen	Positive 16.2.59	Present	Not known	Not done				Not done
13 M*	15.1.59	±22.1.59	Not seen	Not done	Present	Not known	Not done				Not done

C.H. = Coronation Hospital, B.H. = Baragwan th Hospital, N.E.H. = Non-European Hospital.

* Patient died on or about 27 January 195[illegible], without receiving medical attention.

before the onset of other symptoms. Blood counts showed an eosinophilia which ranged from 7% of 9,100 WBCs in a mild case to 63·5% of 24,000 WBCs in one of the most severe. Radiological signs varied from increased bronchovascular markings to bilateral diffuse mottling of the lung fields. Eight patients were passing ascaris ova at the start of the illness and all but one of the remainder had ova in their stools 8 - 16 weeks later. Patz[1] considered that infestation, in some cases probably massive, with *Ascaris lumbricoides* was the most likely cause of this illness. He thought it probable that the patients were infected at the party although he was unable to offer any explanation of how the food or drink had become heavily contaminated with ascaris ova.

In January 1959 we encountered a remarkably similar epidemic in the Ferndale district of Johannesburg. To our knowledge, 13 Bantu adults were involved and the only common factor was that they had all drunk beer from the same source. It is the purpose of this paper to describe the features of this illness and to discuss briefly the possible aetiology.

FEATURES OF THE EPIDEMIC

Thirteen adult Bantu, 5 of whom were females, drank beer from the same source between 15 and 18 January 1959. Most of the witnesses were suspicious of us and their stories were unreliable. It was only by repeated questioning of the 10 whom we were able to contact, and of several relatives or friends who did not drink the beer, that we could ascertain some of the facts. A summary of the relevant features of all the patients is shown in Table I.

Of the 13 patients, 3 (cases 1 - 13) apparently received no medical treatment. One of these (case 13) died on a neighbouring farm on approximately the 7th day of his illness. Another (case 11) was apparently very ill, but was only seen by us some 5 weeks later when he had fully recovered. The third (case 12) was mildly affected. The remaining 10 patients all received medical treatment and our knowledge of their illnesses is fairly complete. Six of them were seen by us personally during the acute stage of the illness and of these 2 were admitted to the Non-European General Hospital (cases 1 and 4), 2 (cases 2 and 3) to Coronation Hospital, 1 (case 5) was examined in her room at Ferndale, and 1 (case 6) was treated by us in the outpatient department of the Non-European General Hospital. Of the remaining 4 patients, 2 (cases 7 and 8) were treated at the Baragwanath Hospital casualty department, 1 (case 9) was treated for 4 days, unfortunately without our knowledge, in the casualty department of the Non-European General Hospital and the last (case 10) received outpatient therapy at Coronation Hospital.

Clinical Features

All patients had developed an itchy skin rash preceding the respiratory symptoms. The rash was still present in 2 (cases 4 and 5) when seen by us and had the appearance of a generalized urticaria.

Respiratory symptoms developed 5 - 7 days after the beer was drunk and consisted of cough and breathlessness with prolonged expiration. The cough was initially dry or productive of a little mucopurulent sputum, but in at least 3 patients (cases 1 - 3) whom we observed, the sputum became frothy, blood-stained and copious 5 - 8 days after the onset of the respiratory symptoms. Five of the 6 patients seen by us (cases 1 - 5) were cyanosed. Dyspnoea was marked in 3 of them (cases 1 - 3) and was moderate in 2 (cases 4 and 5). All 6 had prolonged expiration with rhonchi and crepitations audible over both lung fields. They were pyrexial, the temperature reaching 104° F. in 1 patient (case 2). Similar clinical signs were described in the 4 patients (cases 7 - 10) examined by other medical colleagues. The patients were most ill 5 - 8 days after the onset of respiratory symptoms and about 11 - 14 days after drinking the beer. The man who died (case 13), according to our rather unreliable witnesses, had been coughing and breathless for 5 days. Two patients (1 and 2) developed mild congestive heart failure 6 days after the onset of the respiratory symptoms, with gallop rhythm and electrocardiographic changes compatible with acute cor pulmonale (Fig. 1). Treatment included antibiotics, bronchial antispasmodics, and, in 3 patients (cases 1 - 3), steroids. The 2 patients who developed congestive cardiac failure were digitalized and given mercurial diuretics. None of this therapy seemed to alter the course of the illness. The patients improved spontaneously about 10 days after the onset of respiratory symptoms and all seemed completely well a week later.

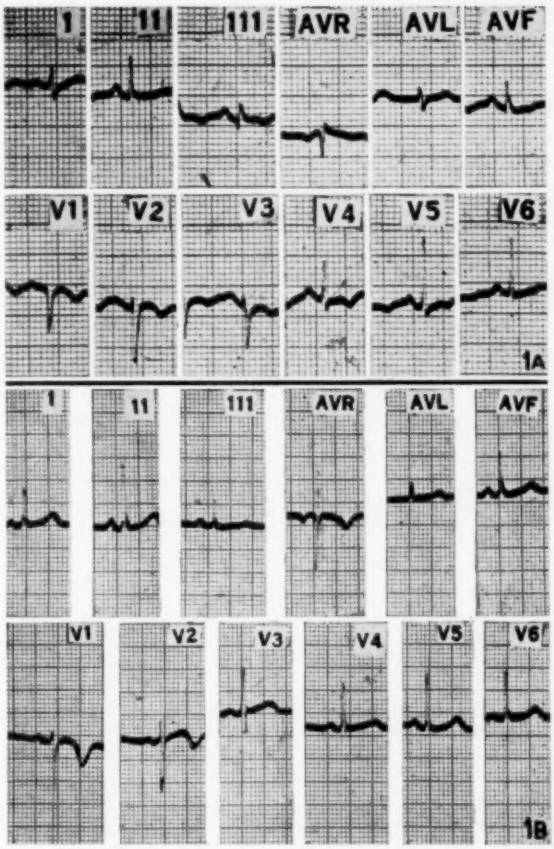

Fig. 1. (*a*) Electrocardiogram of case 2 taken 6 days after the onset of respiratory symptoms when the patient was in mild congestive cardiac failure. The Q in standard lead 3, Qr in AVR, RS in AVL, and inverted or flattened T waves in all the V leads are compatible with acute right ventricular strain.
(*b*) Electrocardiogram 2 weeks later when all symptoms and signs had disappeared. The T waves in V_1 and V_2 are normal for an adult Bantu.[23]

Chest X-rays were taken of 8 patients (cases 1 - 4 and 6 - 9). During the acute stage of the illness, radiological changes varied from increased hilar shadows in the mild cases to extensive mottling of both lung fields in the severe (Fig. 2). Small interlobar effusions were present in 5 cases and probable Kerley 'B' lines in 2. Serial X-rays showed a gradual return to normal within 3 weeks of the onset of the respiratory symptoms.

Special Investigations

Nine patients had at least 1 blood count within 5 weeks of the onset of symptoms. An eosinophilia was present in all

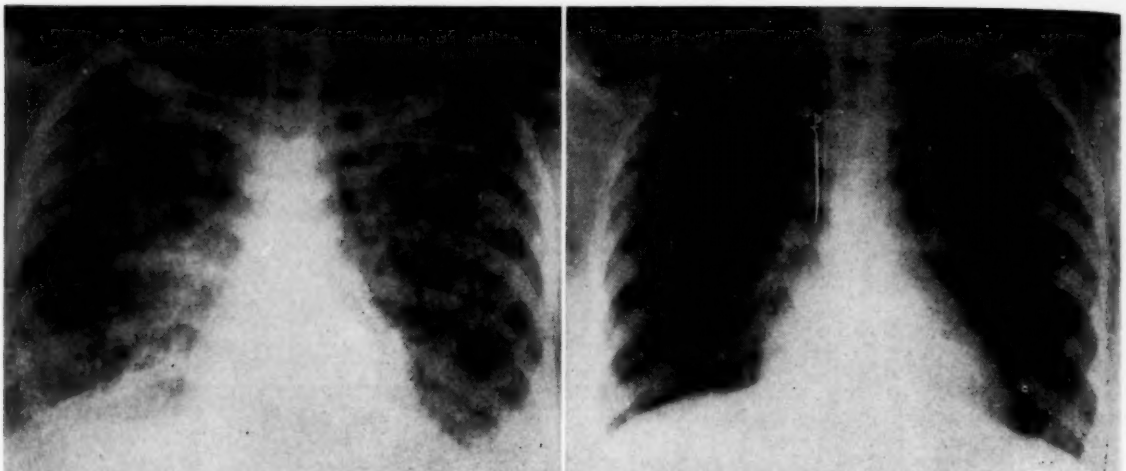

Fig. 2. (*a*) Chest X-ray of case 3 taken 4 days after onset of respiratory symptoms. Extensive bilateral mottling of both lung fields is clearly (*b*) Chest X-ray 8 days later.

and ranged from 7% of 8,900 WBCs to 66% of 20,400 WBCs (Table I). The stools were examined for ascaris ova in all except the man who died (Table I). Seven of the 8 patients whose stools were examined within 4 weeks of the onset of symptoms were passing ascaris ova. Three of the remaining 4 patients were passing ascaris ova when their stools were examined for the first time 4 months later. In 2 patients (cases 1 and 11) we failed to find ova.

The urine of 9 patients was negative for bilharzia ova. The bilharzia complement-fixation test was negative in 8 and doubtful in 2 (cases 5 and 6). The skin test for histoplasmosis was negative in the 8 patients on whom this was performed. Virus studies, including psittacosis, were negative in 4 patients. The sputa of 5 patients (cases 1 - 4 and 6) were sent for microscopic examination and culture. Various, possibly pathogenic, organisms were grown in 4 cases. Nothing of significance was reported on the microscopic examination (performed at the South African Institute for Medical Research) though unfortunately a request for specific search for eosinophils and ascaris larvae had not been made.

DISCUSSION

Ascaris lumbricoides infestation in man starts with the swallowing of embryonated eggs by the host; these eggs then hatch in the small intestine. On the seventh or eighth day after inoculation the larvae migrate in the blood stream through the liver to the lungs where growth continues. They then return to the small intestine, *via* the bronchi, trachea and oesophagus, and reach maturity in 8 - 10 weeks producing unsegmented, non-infective eggs which leave the host in the stools. Further development takes place in a favourable atmosphere, usually moist soil, and within 3 weeks the eggs are infective.[2]

The passage of the larvae through the lungs often produces no symptoms, but radiological signs of transient pulmonary infiltrations may be present at this stage.[2-5] Where respiratory symptoms do occur, however, the clinical syndrome described by Loeffler is the one most frequently seen.[6-11]

Loeffler's syndrome consists of a mild pyrexia lasting about 1 week, associated with a slight productive cough and an eosinophilia. There are minimal signs on clinical examination of the chest, though X-ray examination reveals transient pulmonary infiltrations. Loeffler[7] believed that this syndrome was a manifestation of an allergic reaction to various allergens, but that the migration of ascaris larvae through the lungs was one of the commonest causes. The patient is essentially only slightly ill and spontaneous recovery is invariable.

The condition known as tropical eosinophilia may also be related to some parasitic infestations. This condition, however, which is characterized by pyrexia, weight loss, cough, asthma, radiological signs of pulmonary infiltrations, and a marked eosinophilia, is a more severe illness and runs a protracted course.[12,13] *Toxascaris canis*,[14] mites,[15] and filariae[16] are the parasites most commonly incriminated, and a good response to therapy with organic arsenicals usually occurs.

Pulmonary ascariasis is known to be associated sometimes with a severe acute respiratory illness,[17-21] and the clinical picture is then similar to that described by Patz[1] and to our own cases. In 1922, Koino[19] experimentally swallowed 2,000 mature human ascaris ova. Two days later he developed anorexia and headache, followed 6 days after swallowing the eggs by a severe pyrexial illness with rigors, rapid pulse, cyanosis, extreme dyspnoea, and paroxysmal cough productive of increasing quantities of sputum which later became blood stained. Râles were present throughout the chest and ascaris larvae were recovered from the sputum. The illness lasted 10 days. Beaver and Danaraj[20] described an adult male who presented with a 10-day history of very severe bronchial asthma. On examination the patient was critically ill, coughing, and very dyspnoeic with marked bronchospasm. A blood count showed a leucocytosis of 21,400 with 37% eosinophils. The patient died a few hours after admission; at autopsy there was marked dilatation of the right ven-

tricle and ascaris larvae were found in the bronchioles. Hemming[21] observed that severe respiratory symptoms were common among malnourished Indian children in the Fiji Islands. Within a period of only 40 days he saw 100 cases of 'ascaris pneumonitis'. His patients, one of whom died, were all seriously ill and had presented with severe cough, asthma, cyanosis and pyrexia.

The epidemic we have described seems to us virtually identical to that reported previously by Patz.[1] The illness itself, including the urticaria, small interlobar effusions, eosinophilia and severe asthma, is strongly suggestive of an acute allergic reaction and it seems likely that the causative agent was contained in the beer. Certainly everyone who drank this particular brew became ill to a greater or lesser extent, whereas at least 1 person, to our knowledge, who was present at the party but did not drink, remained completely well. Neither Patz[1] nor ourselves, however, were able to ascertain the exact constituents of the beer, and in neither instance is there conclusive evidence that the swallowing of ascaris ova was the cause of the subsequent illness. Possibly due to the death of one of their number, those who had attended the party were suspicious, particularly the man Arthur (case 1) and his wife who brewed the beer. Originally, all the witnesses had denied that there was anything unusual about the beer, but after repeated questioning and some direct suggestion by us, several of them later volunteered that it had contained sand. Since the beer had been hidden for 3 days on the bank of a nearby stream in order to avoid detection by the police, it might have been contaminated accidentally or by someone ill-disposed towards the brewers. Perhaps another possibility is that it was made originally with impure river water.

Of the 12 cases described by Patz,[1] 8 had ova in the stools at the start of the illness and were thus almost certainly already infected when they drank the beer. Three of his remaining 4 patients had ova in the stools 8 - 16 weeks later. Patz[1] considered that the patients already infected had a higher eosinophilia and more severe illness than the others. Since it usually takes at least 8 weeks for the worm to reach maturity and therefore produce eggs,[2] however, it is debatable whether his 3 patients who had ova in their stools at 8 - 16 weeks were not also already infected at the time of the beer drink. Of our 13 cases, the man who died was never examined, 7 patients had ova when the stools were examined 4 - 5 weeks after drinking the beer, and 3 others had ova present 3 months later. The probable existence of intestinal ascaris infestation in the majority of Patz's and our patients at the time of the respective 'parties', when infection or re-infection is thought to have occurred, does not weaken the argument that the acute pulmonary illness could also have been due to ascariasis. Beaver and Danaraj,[20] and Baumann,[8] believe that the most severe pulmonary reactions occur either in patients already infested with ascaris, and consequently more liable to be allergic to the larvae passing through the lungs, or in those where a massive infestation takes place for the first time.

In 2 patients (cases 1 and 11), one of whom had been extremely ill (case 1), we were unable to find ova at any time. Absence of ova in the stools, however, is not incompatible with ascaris infestation.[3,9,10,22] Minteer *et al.*[22] described 11 patients in whom adult ascaris worms were demonstrated by barium-meal examination, yet only 3 had positive stools. In their remaining 8 patients, no ova were found in a total of 20 stool examinations.

We are unable to offer any alternative explanation for the cause of this epidemic. The only common factor to all our patients was that they drank beer from the same source. Four of them (cases 4, 7, 8 and 10) bought the beer and drank it elsewhere, and at least 1 person, in fact the wife of Arthur (case 1), who was present at the party but did not drink, remained well.

If an allergic reaction to the passage of ascaris larvae through the lungs was the cause, however, then it is surprising that this syndrome is not seen more frequently. Infestation with ascaris is extremely common in this country especially among the Bantu population. A possible explanation is that our patients swallowed an unusually large number of ova and, especially as at least 7 of them were already infected, were probably more liable to have a severe reaction.[5,20] How or why the beer became contaminated with these ova remains a problem unsolved by us.

SUMMARY

An epidemic is described of an acute respiratory illness in 13 adult Bantu (5 females) which occurred during January 1959 in the Ferndale district of Johannesburg. Features of the illness included a generalized urticarial skin rash, cough, breathlessness with prolonged expiration, cyanosis and pyrexia. Two patients developed congestive cardiac failure with electrocardiographic evidence of acute cor pulmonale. Radiological changes varied from increased hilar shadows in the mild cases to extensive mottling of both lung fields in the severe. An eosinophilia was present in all 9 patients on whom a blood count was done. The stools of 12 patients were examined for ascaris ova and were positive in 10. One patient died without receiving medical attention.

This epidemic is thought to be remarkably similar to that reported previously by Patz,[1] which took place during November 1956 in 12 Bantu males at Middelburg, Transvaal, and which was attributed to infestation with *Ascaris lumbricoides*.

The common factor to our patients was that they had all drunk beer from the same source. It is postulated that the respiratory illness resulted from infestation with *Ascaris lumbricoides* and that the beer was the source of infection.

The pulmonary manifestations of ascariasis are briefly discussed.

ADDENDUM

Since this paper was written, a case of pulmonary ascariasis was reported by Simson and Heinz.[24] The patient, a 3-year-old Bantu female, died after a severe respiratory illness of four days' duration. On histological examination of the lungs, numerous nematode larvae, resembling *Ascaris lumbricoides*, were found in the bronchioles. As in our cases, the source of the infestation was unknown.

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‚DIE EER EN BELANGE VAN DIE MEDIESE BEROEP'*

J. W. VAN DER RIET, *President, Tak Oranje-Vrystaat en Basoetoland, (M.V.S.A.)*, 1960

Dit is nou twee jaar sedert ek deur u aangewys is as die President-Voorsitter van die Tak Oranje-Vrystaat en Basoetoland en, alhoewel hierdie 'n uittreerede is, wil ek graag gebruik maak van die geleentheid om u weer eens te bedank vir die voorreg en die eer wat u my aangedoen het. Die jaar wat ek as Voorsitter gedien het, het nie juis enige hoogtepunt bereik nie, maar dit het heel waarskynlik 'n keerpunt in die mediese praktyk gebring. Siekteversekering en veral vooraf betaalde siektedekking, soos deur versekeringsmaatskappye aangebied word, is die saak wat ons as individue, en die Vereniging in sy geheel, besig gehou het. Finansiële sake het die verrigtinge en die werksaamhede van die Federale Raad oorheers. Alhoewel dit noodsaaklik is dat ons georganiseerd is en ons geldsake nugter benader, is dit my mening dat die eerste deel van die artikel in die Akte van Oprigting van die Mediese Vereniging van Suid-Afrika weer meer beklemtoon moet word, naamlik: ‚Om die geneeskunde en verwante wetenskappe te bevorder, en om die eer en die belange van die mediese beroep te handhaaf en te beskerm'. Ek wil dus in the eerste instansie ons professie in oënskou neem en vir 'n oomblik die vraag stel (en probeer beantwoord) of die belange van die geneesheer deur die jare, of hy nou ook al 'n lid is van die Mediese Vereniging of nie, en of hy hier of elders in die wêreld woon, wel in eer gehou is? Die tweede vraag wat ek wil stel is hoe die geneesheer daardie voorreg verwerf het. Eindelik wil ek die vraag ondersoek van hoe die professie sy ereposisie kan handhaaf en beskerm.

Dr. v. d. Riet

Die Eer van die Mediese Beroep

Navorsing toon dat siekte reeds in prehistoriese tye, tesame met die eerste tekens van lewe, teenwoordig was — selfs soos ons dit vandag ken, bv. osteitis, gewrigsontsteking, ens. Deur die verskillende fases van mediese praktyk, die mistiese, die goddelike, (soos vertolk deur die Griekse mitologie tot by die vader van moderne medisyne, nl. Hippokrates) was die beoefening van dié kuns altyd toevertrou aan iemand met spesiale eienskappe. Dié persoon het dan ook altyd 'n verhewe posisie beklee.

Die gesogte ere-posisie en status wat geneeshere beklee het, is te danke aan hulle karakter, toewyding, en besondere kennis — kennis wat hulle in die nastrewe van hulle praktyk en deur die intieme omgang met die mens in sy swakheid en ellende opgedoen het, en ook, en veral, besondere vakkundige kennis. As gevolg van hierdie opvoedkundige agtergrond, tree die geneesheer as raadgewer op, nie alleen vir sy gewone pasiënte nie, maar dikwels ook vir vorste en regeerders.

In teenstelling hiermee wil ek dit graag noem dat waar die mediese professie verkleineer word, dit altyd oor die een of ander geldelike implikasie is. Alhoewel hierdie aspek vir ons as geneeshere net so belangrik is as vir enige ander burger, as verantwoordelike versorger van afhanklikes, wil ek hierdie sy van die saak met al sy fasette liewer links laat lê.

Hoe het die geneesheer sy ere-posisie verwerf?

In hierdie verband wil ek weer na die geskiedenis verwys. In die dae van die primitiewe mens was dit moontlik om sekere siektetoestande te vereenselwig met fisiese oorsake, maar in baie gevalle was die siekte aan iets bonatuurliks toegeskryf. Dit het gunstige omstandighede vir die toordokter geskep — iemand wat op die bomenslike vermoë aanspraak gemaak het om met die bonatuurlike elemente in verbinding te tree, en sodoende groot teenspoed en rampe te verhoed. Voorbeelde hiervan vind ons vandag nog onder primitiewe rasse.

Nader aan vandag (as 'n mens die verhaal van die groot beskawings lees), vind ons dat daar in die Egiptiese beskawing die eerste bewyse van 'n georganiseerde mediese klas is. Lede van dié klas het spesiale voorregte en titels ontvang om hulle as verhewe priesters in 'n klas van hulle eie te plaas. Dit is van hulle wat Homer in sy Odyssee skryf ‚each is a physician with knowledge beyond all men'. In die vroeë Griekse tye was die vermoeë om te genees in die hande van die gode, maar later vind ons die uitstaande figuur van Hippokrates — dié persoonlikheid aan wie die nuwe fase van mediese denkwyse en praktyk gekoppel word. Hy lui die wetenskaplike benadering tot die kuns van mediese praktyk in. Medisyne word van nou af gebaseer op 'n kennis van die natuurwetenskappe, waarneming van die pasiënt, 'n grondige kennis van praktiese medisyne, en 'n heldere beredenering oor die verhouding van oorsaak en gevolge, gegrond op etiese begrippe van morele wette.

In Rome was daar neergesien op die geneesheer, maar met die latere Griekse invloed op Rome, vind ons dat die beter toegeruste Griekse geneesheer met sy kennis baie gou die antagonisme wat daar geheers het, laat verdwyn het, en geneeshere is toegelaat tot die hoogste kringe van die samelewing.

Sonder om die geskiedkundige agtergrond van die saak onnodig te beklemtoon, dink mens onwillekeurig aan die voortbouing op die Hippokratiese fondamente: en aan manne soos Leonardo da Vinci en Vesalius wat die grondslag gelê het vir die basiese wetenskappe van ons hedendaagse mediese studie. In hierdie verband moet daar ook gedink word aan persone soos Morgangi, en al die

* Afskeidsrede, Bloemfontein, 4 Maart 1961.

groot wetenskaplike navorsers en kliniese genieë. Elke land het sy groot figure gelewer wat hulle bydraes gelewer het tot die mediese wetenskap van vandag. Die nuwe ontdekkings wat toen ter tyd spesialis kennis behels het, het mettertyd die algemene kennis van die gewone geneesheer geword.

In die twintigste eeu met sy sosio-ekonomiese veranderinge, verbindinge, en verspreiding van kennis, het die proses van nuwe ontdekkings al vinniger geword. Die verwante wetenskappe het so 'n fenomenale uitbreiding ondergaan, dat dit haas onmoontlik vir een man geword het om 'n deeglike kennis van die hele mediese wetenskap te bemeester. Vir die geneesheer om sy status te handhaaf en sy ere-posisie, wat op kennis gebaseer is, te behou, is dit dus geen wonder dat spesialisasie sulke afmetings aanneem nie.

Hierdie neiging is myns insiens in ooreenstemming met die professie se hoogste ideale, mits dit nie uit 'n winsmotief of uit gemaksugtigheid gedoen word nie. Daar is egter nie 'n meer stimulerende invloed op 'n mens nie as om 'n moderne mediese skool te besoek waar die strewe na kennis voorrang geniet, en waar elkeen sy taak, as 'n diens aan sy medemens, met ywer en lus vervul — selde teen 'n lonende vergoeding.

Hoe kan die eer van die mediese beroep gehandhaaf word?

Ek laat die kwessie van voorgraadse opleiding vir die doel van hierdie rede buite rekening, en handel daardie aspek af deur die algemene stelling dat die toekomstige geneesheer moet toegerus wees met kennis van die heel nuutste ontwikkelinge. Die kennis moet gegrond wees op kennis van die basiese vakke, en die nuwe wendings in kliniese vakke moet konserwatief genoeg voorgedra word om die jong geneesheer die nodige perspektief te gee. Dwarsdeur die mediese wêreld word spesiale aandag aan studieplanne gewy, veral op die gebied van nagraadse studie. In Amerika, waar die meeste fasiliteite beskikbaar is en ontwikkeling in die hoogste rat is, word planne beraam om die geweldige gespesialiseerde kennis tot die grootste voordeel vir die mensdom aan te wend. Deur ultra-spesialisasie het hulle die belangrike skakel egter geëlimineer, nl. die ou familiedokter of algemene praktisyn. In die nuutste skrywe wat ek in hierdie verband gelees het, word daar gedink aan die toepassing van die sogenaamde Y plan, om 'n nuwe soort spesialis, die ‚family practitioner', op te lei. Hy moet dien as skakel tussen die pasiënt en die super-spesialis.

Suid-Afrika is onderworpe aan dieselfde invloede wat van toepassing is op die buiteland, maar die stelsel van praktyk soos ons dit ken, sal nog baie jare moet voortduur. Afstande en die yl bevolking is maar twee faktore wat die algemene praktisyn op die platteland onmisbaar maak. In die stede vervul hy sy taak goed en dien daar ook as 'n onmisbare skakel.

Om hom egter in beide plekke in sy ere-posisie te handhaaf, is dit gebiedend noodsaaklik dat daar georganiseerde pogings gemaak word om hom in voeling met die ontwikkeling van die wetenskappe te hou.

Ek wil my dus nou bepaal tot die middel wat na my mening noodsaaklik is vir die handhawing en beskerming van die geneesheer se ere-posisie, nl. nagraadse studie.

Onder geneesheer sluit ek beide die spesialis en die algemene praktisyn in. Spesialisasie is die bekwaam-making van die indiwidu vir die beoefening van 'n sekere aspek van die geneeskunde, maar sodra die betrokke geneesheer in die praktyk kom, moet hy dieselfde mate van opknapping geniet as sy kollega, die algemene praktisyn, al sou dit in graad verskil.

Die universiteite is op die oomblik ingestel op voorgraadse studie, en alhoewel hulle fasiliteite vir spesialisasie bied, is daar nie voldoende geleenthede vir die praktisyn wat sy kennis wil verfris, om dit te doen somder om noodwendig te spesialiseer nie.

Twee organisasies wat hulle ten doel stel om in hierdie leemte te voorsien is die Kollege van Interniste, Chirurge en Ginekoloë van Suid-Afrika, en die Kollege van Algemene Praktisyns. Laasgenoemde is nog nie onafhanklik van die Britse kollege nie, maar sal binnekort sy eie Suid-Afrikaanse Raad verkry. Ek vind dit jammer dat die algemene praktisyns nie as 'n selfstandige groep deur die Kollege van Interniste, Chirurge en Ginekoloë van Suid-Afrika erken kan word en in die organisasie as groep met sy eie identiteit opgeneem kan word nie.

Dieselfde mening geld vir die baie groepe binne die Mediese Vereniging van Suid-Afrika. Elkeen het sy eie probleme en wil graag sy eie vak bevorder, en dit vind ek goed, binne die raamwerk van die groter organisasie. Maar dit moet nie verbrokkeling in die hand werk nie.

Soos blyk uit die verslag van die Nagraadse Komitee[1] het ons ver gevorder en sal Bloemfontein binne afsienbare tyd die nodige fasiliteite vir die opleiding in die basiese vakke kan bied.

Met die samewerking van die Universiteit van die Oranje-Vrystaat is dit ons voorneme om 'n akademiese standaard te handhaaf. As sake vorder soos ons verwag, sal Bloemfontein nog sy Geneeskundige fakulteit verkry.

Intussen wil ek die kollegas van die platteland nooi om by die algemene praktisynsgroep aan te sluit, en terselfdertyd by die plaaslike fakulteit van die Kollege van Algemene Praktisyns. Die kern van hierdie saak is reeds in werking, en ons soek uitbreiding. Kliniese vergaderings van hoë gehalte het nou 'n instelling geword, en word elke tweede Maandagaand gehou. Dit voldoen in ruim mate aan die vereistes van die Kollege.

As genoeg ondersteuning verkry word, kan addisionele aantreklike en leersame programme opgestel word. Ander ondernemings soos, bv. kliniese navorsing deur verskeie kollegas in verskeie dele van die Vrystaat, kan aangedurf word. Dit is reeds 'n instelling in Brittanje. Dit sal 'n lewende belang by die kollegas kweek, en dit sal ons vrywaar teen propagandistiese kennis. Terselfdertyd kan ons daardeur 'n landsdiens verrig en sodoende onnodige uitgawe aan oortollige invoer-goedere en duur medisynes, verhoed. Ander probleme mag ook op so 'n wyse ondersoek word bv. padongelukke.

Ek wil dus die mening uitspreek dat die mediese professie sy eer voortaan, soos in die verlede, alleen sal kan handhaaf en beskerm deur die standaard van sy kennis hoog te hou, en dit kan slegs deur 'n lewende belangstelling en voortdurende studie van die verskeie vertakkinge van die geneeskunde en verwante wetenskappe verkry word.

1. Van der Riet (1961): S. Afr. T. Geneesk., 35, 296.

AMPTELIKE AANKONDIGING : OFFICIAL ANNOUNCEMENTS

TARIEF VIR GOEDGEKEURDE MEDIESE HULPVERENIGINGS

Die aandag van lede van die Vereniging word bepaal by die eerste paragraaf van klousule 2 van die Algemene Inleiding tot die Tarief waarin geneeshere gewaarsku word dat hulle met die betrokke mediese hulpvereniging in verbinding moet tree alvorens hulle 'n duur behandeling of operasie van een van die lede daarvan onderneem.

Dit is gebiedend noodsaaklik dat hierdie instruksie uitgevoer word om verleentheid en verlies van gelde te vermy, indien daar gevind word dat die hulpvereniging om verskeie redes nie verantwoordelikheid vir die rekening van die lid kan aanvaar nie.

L. M. Marchand
Medesekretaris

Plaza-gebou 28
Pretoria
27 April 1961

TARIFF OF FEES FOR APPROVED MEDICAL AID SOCIETIES

The attention of members of the Association is drawn to the first paragraph of clause 2 of the General Preamble to the Tariff, in which practitioners are warned to communicate with the medical aid society concerned before undertaking a costly procedure or medical service for one of its members.

It is imperative that practitioners should follow this instruction in order to avoid embarrassment and loss of fees if it is found that, for various reasons, the society cannot accept liability for the account of the member.

L. M. Marchand
Associate Secretary

28 Plaza Building
Pretoria
27 April 1961

MEDIESE HULPVERENIGINGS

Op sy vergadering op 21 April 1961 te Pretoria gehou, het die Uitvoerende Komitee van die Federale Raad die onderstaande nuwe mediese hulpverenigings goedgekeur. Die goedkeuring tree van 1 Mei 1961 af in werking:

1. Alliance Assurance Co. Ltd. Medical Aid Society, Posbus 635, Kaapstad.
2. Bunzl Paper (S.A.) Medical Aid Society, Posbus 2, Maitland, Kp.
3. D.I.S.W. (Dunswart Iron and Steel Works) Medical Aid Society, Posbus 290, Benoni.
4. Irvine Chapman Medical Aid Scheme, Posbus 316, Vereeniging.
5. Reunert and Lenz Ltd. Medical Aid Society, Posbus 92, Johannesburg.
6. Sanlam Kantoorpersoneel Siektefonds, Posbus 1, Sanlamhof, Kp.

Die name van die volgende hulpverenigings moet van die lys van goedgekeurde hulpverenigings geskrap word:

Standard Brass Medical Aid Society, Posbus 229, Benoni.

Begbie Medical Benefit Fund, Posbus 192, Middelburg, Transvaal.

Geliewe te let op die verandering van naam van die volgende hulpverenigings:

African Homes Trust Sick Fund na ‚Homes Trust Sick Fund'.

Polliack Group Medical Aid Society na ‚Bothner Group Medical Aid Society'.

'n Volledige lys van die goedgekeurde mediese hulpverenigings sal in die volgende uitgawe van die *Tydskrif* verskyn.

L. M. Marchand
Medesekretaris

Plaza-gebou 28
Pretoria
26 April 1961

MEDICAL AID SOCIETIES

The following new medical aid societies were approved by the Executive Committee of Federal Council at its meeting held at Pretoria on 21 April 1961. The approval takes effect from 1 May 1961:

1. Alliance Assurance Co. Ltd. Medical Aid Society, P.O. Box 635, Cape Town.
2. Bunzl Paper (S.A.) Medical Aid Society, P.O. Box 2, Maitland, C.P.
3. D.I.S.W. (Dunswart Iron and Steel Works) Medical Aid Society, P.O. Box 290, Benoni.
4. Irvine Chapman Medical Aid Scheme, P.O. Box 316, Vereeniging.
5. Reunert and Lenz Ltd. Medical Aid Society, P.O. Box 92, Johannesburg.
6. Sanlam Kantoorpersoneel Siektefonds, P.O. Box 1, Sanlamhof, C.P.

The names of the following medical aid societies must be removed from the list of approved societies:

Standard Brass Medical Aid Society, P.O. Box 229, Benoni.

Begbie Medical Benefit Fund, P.O. Box 192, Middelburg, Transvaal.

Kindly note the change of name of the following societies:

African Homes Trust Sick Fund to 'Homes Trust Sick Fund'.

Polliack Group Medical Aid Society to 'Bothner Group Medical Aid Society'.

A complete list of the approved medical aid societies will be published in the following issue of the *Journal*.

L. M. Marchand
Associate Secretary

28 Plaza Building
Pretoria
26 April 1961

DIE LIEFDADIGHEIDSFONDS : THE BENEVOLENT FUND

Met dank word die volgende skenkings gedurende die maand April 1961 erken:
The following donations during April 1961 are gratefully acknowledged:

Geloftekaarte ter Nagedagtenis aan: Votive cards in Memory of:

Miss Peck Lam *by* Dr. H. A. Kalley; Mrs. Kate Capley *by* Natal Coastal Branch (M.A.S.A.); Dr. R. A. Moore-Dyke and Dr. S. Per *by* South African Society of Anaesthetists; Dr. D. A. S. Sichel *by* Dr. A. W. Sichel and Drs. Grant-Whyte and partners.

Totaal Ontvang van Geloftekaarte:
Total Received from Votive Cards: R20.95

Dienste Gelewer aan: Services Rendered to:

Rev. E. H. and Mrs. Smithens *by* Drs. R. Gillman, H. Sandeman, D. Black and D. S. Hawk.

Dr. M. Fouche *by* Mr. V. A. Douglas, Mr. A. Leonsins, Miss P. Knocker, Mr. P. Theron, Prof. V. Gear, Drs. L. Denis-Lester, B. Bradlow, R. Sauer, D. R. Morris, J. Gear, N. de La Hunt, J. Durham, H. Gliekenberg, R. Glyn Thomas, Sims, Gluckman, Lewin, Bloomberg, Helman, and the Director and staff of the South African Institute for Medical Research.

Mev. C. Gobregts *deur* Prof. J. de Villiers.

Dr. L. M. Oosthuizen *deur* Drs. L. Lane en J. T. Russel.

Mrs. F. P. S. le Roux *by* Dr. W. H. Lawrance.

Dr. and Mrs. E. J. Dyke *by* Drs. A. J. Biesman Simons, A. Kessler, J. Lee, and M. J. Viljoen.

Dr. A. J. Orenstein *by* Mr. J. A. Douglas, and Dr. F. J. Durham.

Jo-Ann, daughter of Dr. H. Loewenstein *by* Dr. W. A. Lombard.

Totaal Ontvang van Dienste Gelewer:
Total Received from Services Rendered: R438.70

Skenkings: Donations:

Drs. Harries, Kuschke, Hofmeyr, E. T. Dietrich, C. T. Koster, J. O. Harle, D. L. Ranking, H. Levin, J. R. v. Heerden, J. L. Parker, E. Frankenfeld, W. F. Leith, J. E. Rea, M. G. Erasmus, R. Conley, C. D. Scott, E. Wyngaard, P. S. Grove, H. H. Stormanns, M. A. Lloyd, C. L. Lauf, P. du Toit, and S. Donen.

	R
Total	25.25
Dr. T. S. Eddy	4.00

Totaal Ontvang van Skenkings:
Total Received from Donations: R29.25

Groot Totaal: Grand Total R488.90

WORLD LIST OF FUTURE INTERNATIONAL MEETINGS

ALTERATIONS AND ADDITIONS NOTIFIED DURING APRIL 1961

French Congress of Anesthesiology, Nancy, 18 - 21 May 1961. Dr. J. M. Picard, Hôpital Central, Av. de Lattre-de-Tassigny, Nancy, France.

International Society for Clinical and Experimental Hypnosis, Rio de Janeiro, 16 - 22 July 1961. 33 East 65th Street, New York 21, N.Y.

International Conference on Congenital Malformations, Vienna, July/August 1961. Dr. Bowman, Pineland Hospital, Pownal, Me.

Inter-American Conference on Occupational Medicine and Toxicology, Miami, 6 - 10 August 1961. Dr. W. B. Deichmann, School of Medicine, University of Miami, Coral Gables, Fla.

International Congress on Mental Health, Paris, 30 August-5 September 1961. Secretary General, World Federation for Mental Health, 19 Manchester Street, London, W. 1.

International Congress of Neurological Surgery, Washington, 14 - 20 October 1961. Secretary General, 525 East 68th Street, New York 21, N.Y.

International Congress of Allergology, New York, 15 - 22 October 1961. Dr. W. B. Sherman, 60 East 58th Street, New York 22, N.Y.

UNIVERSITEITSNUUS : UNIVERSITY NEWS

INSTELLING VAN PH.D.-GRAAD (GENEESKUNDE)

Die Universiteitsraad van Stellenbosch het op sy jongste vergadering besluit om, benewens die M.D.-graad, ook 'n Ph.D.-graad (geneeskunde) in die Fakulteit van Geneeskunde in te stel. 'n Kandidaat kan tot die graad van Doktor in die Wysbegeerte (Geneeskunde) toegelaat word:

1. Vier jaar nadat hy die Baccalaureusgrade in die geneeskunde en in die snykunde van die Universiteit verwerf het, of nadat hy 'n ander kwalifikasie verwerf het wat na die oordeel van die Senaat van 'n voldoende standaard is; en

2. mits hy vir ten minste een jaar vir die graad van Doktor in die Wysbegeerte (Geneeskunde) ingeskryf was.

Die graad Ph. D. (Med.) sal nie vir registrasie as spesialis by die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad kan dien nie.

IN DIE VERBYGAAN : PASSING EVENTS

University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.), Joint Lectures. The next lecture in this series will be held on Wednesday 17 May at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Dr. H. van Diggelen will speak on 'Principles of physiotherapy'. All members of the Medical Association are welcome to attend this lecture.

* * *

Research Forum, University of Cape Town. A meeting of Research Forum will be held on Thursday 18 May at 4 p.m. in the Tutorial Room of the Pathology Department, Medical School, Observatory, Cape. Dr. J. Herman will speak on 'Gout and diabetes'. All those interested are invited to attend this meeting.

* * *

Hart-long Groep van die Universiteit van Stellenbosch. Die volgende vergadering van die Groep word gehou op Donderdag 18 Mei 1961 om 8 nm. in die Groot Voorlesingsaal, Kliniese-gebou, Karl Bremer-hospitaal, Bellville. Prof. A. J. Brink sal optree as spreker oor 'Atrium septum defek'. Alle belangstellendes word vriendelik uitgenooi om die vergadering by te woon.

* * *

Dr. Ellis Cooper has commenced practice as an obstetrician and gynaecologist at 1105 Ingrams Building, Twist Street, Hillbrow, Johannesburg. Telephones: Rooms 449235, residence 413513.

* * *

The South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 22 May at 5.10 p.m. in the Institute Lecture Theatre. Dr. S. M. Lewis will speak on 'Paroxysmal nocturnal haemoglobinuria'.

Smith, Kline and French Laboratories Award for Postgraduate Clinical Study in South Africa: 1961 Fellowship. This award has been established by a grant from SKF Laboratories (Pty.) Limited, P.O. Box 784, Port Elizabeth. This is the South African branch of Smith, Kline and French Laboratories Ltd., London.

The Selection Committee (an entirely independent board of medical practitioners) consists of the following: Prof. J. F. Brock, Prof. J. H. Louw (Cape Town); Prof. G. A. Elliott, Prof. E. H. Cluver, Dr. H. A. Shapiro (Hon. Chairman), Dr. M. Shapiro, Dr. M. M. Suzman (Johannesburg), and Prof. H. W. Snyman (Pretoria).

Applications are invited from registered *general practitioners* who have been in active practice in South Africa for at least 7 years. The Bursary is intended for postgraduate clinical study and not for medical research. It is available for not less than a 2-month period at any medical school in South Africa. The total value of the Bursary is R600.

The candidate must submit a brief statement of his proposed course of study, and indicate the institution at which he intends to undertake it. No payments will be disbursed to the successful applicant until he has satisfied the Selection Committee that he has been accepted for the period of postgraduate study at a South African medical school. Applications must be made on the prescribed forms which are available from Dr. H. A. Shapiro, Selection Committee, SKF Laboratories Award for Postgraduate Clinical Study, P. O. Box 1010, Johannesburg. The closing date for applications is 30 June 1961.

* * *

Somerset Hospital Monthly Clinical Evening will be held on 16 May at 8.15 p.m. at the Somerset Hospital. These meetings are held regularly on the third Tuesday of each month.

NUWE PREPARATE EN TOESTELLE : NEW PREPARATIONS AND APPLIANCES

ENAVID—NEW DOSAGES AND NEW-STRENGTH TABLET

The revised dosages which have been evolved during extensive clinical trials lasting over 5 years, have necessitated the introduction of an Enavid 5 mg. tablet.

The simplified dosages now mean that most gynaecological disorders are treated with Enavid, many of them with the same dosage schedule.

For example, menorrhagia, irregular menstruation, dysmenorrhoea, premenstrual tension, amenorrhoea, oligomenorrhoea are all controlled by one 5 mg. tablet, administered from day 5 for 20 days in each of three menstrual cycles. This same dosage prevents ovulation during the treated cycles.

Details of the new dosage for metrorrhagia, advancement or postponement of menstruation, endometriosis, threatened or habitual abortions and infertility due to inadequate luteal phase, are available on request from Keatings Pharmaceuticals Ltd., P.O. Box 256, Johannesburg.

CIROTYL

Parke Davis Laboratories (Pty.) Ltd. announce the introduction of Cirotyl, and supply the following information:

The addition of a wetting agent and faecal softener to the formula of Cirotyl makes this liquid laxative doubly effective in that it combines several desirable features in one product.

Description. Cirotyl is a cherry-flavoured, liquid *dual-action* laxative preparation containing as its active constituents diacetoxydiphenylisatin and propylene oxide ethylene oxide polymer. Diacetoxydiphenylisatin is a synthetic form of the active principle of prunes; its gentle but persistent action mildly stimulates normal peristalsis.

The propylene oxide ethylene oxide polymer is a wetting agent and faecal softener, which acts as a surface-tension depressant permitting fluids in the colon to penetrate into, and soften, hard, dry faecal matter. This results in a softer and more homogeneous stool which is then easily evacuated, aided by the mild peristaltic action of diacetoxydiphenylisatin. Cirotyl counteracts intestinal atonicity through a double mechanism; by direct chemical stimulus of the mucosa and by reflex stimulus through absorption and retention of fluids in the faeces.

Indications. Cirotyl is indicated for the prevention and treatment of occasional or habitual constipation and in cases where it is necessary to maintain the stool soft, as in pre- and postsurgical patients, in cases of anal fissure, rectal abscesses or haemorrhoids, in pregnancy and convalescence, in invalids, geriatric and sedentary patients or patients confined to bed for long periods, in paralysis or muscular weakness, and in patients with cardiovascular disease.

Dosage and administration. The recommended initial dosage is: Infants, ½ - ½ teaspoonful; children 1 - 6 years, ½ - 1 teaspoonful; children above 6 years and adolescents, 1 teaspoonful; adults, 2 teaspoonfuls.

Water should be taken after each dose, which should be ad-

ministered at bedtime or in the early morning before breakfast. As the optimal dose varies from patient to patient according to the degree of constipation, the dosage schedule should be adjusted to fit each individual case.

Presentation. Cirotyl is supplied in bottles of 4 fl. ozs., each teaspoonful (5 ml.) of the suspension containing 2 mg. of diacetoxydiphenylisatin and 100 mg. of propylene oxide ethylene oxide polymer.

Further information is obtainable from Parke, Davis Laboratories (Pty.) Ltd., P.O. Box 24, Isando, Transvaal.

BOEKBESPREKINGS : BOOK REVIEWS

CLINICAL MEDICINE

Clinical Medicine — The Modern Approach. By A. E. Clark-Kennedy and C. W. Bartley. R2.50. London: Pitman Medical. 1960.

The authors have set out to provide a book for use by the undergraduate student who is starting clinical medicine for the first time. No teacher of medicine will deny the need for such a work. The present-day undergraduate student, starting his clinical years, has excellent systematic textbooks available to him and, over the course of two or three years, receives formal and informal tuition covering the whole field of medicine. However, neither source of information meets the needs of the beginner. The textbook is too indigestible except as a source of reference for specific diseases, and tuition cannot cover the whole subject fast enough in the early stages. The authors have tried to fill this need by producing a readable and relatively short book containing a 'bird's eye view' of the whole subject of clinical medicine.

For over three decades Dr. A. E. Clark-Kennedy has divided his life between long weekends as fellow of Corpus Christi College, Cambridge and tutor in physiology to generations of Cambridge medical undergraduates, and mid-weeks devoted to his functions as physician to the London Hospital, consulting physician and Dean of the London Hospital Medical School. His lively and provocative mind has been a stimulus to generations of Cambridge and London Hospital medical students. In his approaching retirement he has combined with Dr. C. W. Bartley to record his philosophy on the wider or modern approach to clinical medicine.

The book opens with an introductory note on how to use it. Part I then discusses 'The patient and his disease', a broad philosophical treatment of the aetiology of disease with the emphasis on constitution, environment, and mind-body inter-relationship in the production of disordered function. Many teachers will hesitate to offer so many teleological explanations for physiological and pathological processes. Parts II and III deal with functional and organic disorders respectively, together forming the longest section of the book. Under a few broad aetiological headings the whole field of human disease is covered. To make the text flow more easily, separate diseases are not given sub-titles, but the name of the disease is printed in heavier type the first time it is mentioned. The effect is very satisfactory. A heavy-type title and the brief account that follows usually stands by itself, out of context, as an adequate summary. In order to present a picture of disease as a whole, surgical and other 'non-medical' conditions are also included in this section.

Part IV deals with 'Clinical diagnosis'. On the whole the discussion of signs and symptoms is excellent, but, of necessity, the condensation of this large subject into such a short space results in broad generalizations and simplifications at the expense of scientific accuracy, especially when biochemical phenomena are discussed. The same holds true for Part V: 'Principles of prevention and treatment'. In this, as in other sections of the book, the psychoses and psychoneuroses are dealt with particularly well.

On the whole, the authors appear to have achieved their object of providing a 'bird's eye view' of clinical medicine for the beginner. The printing and binding of the volume are excellent and the price very modest.

J.F.B. and H.L.F.C.

RADIATION INJURY

Radiation Injury in Man. By Eugene P. Cronkite, M.D. and Victor P. Bond, M.D., Ph.D. Pp. vii+200. R5.20 net. Oxford: Blackwell Scientific Publications Ltd. 1960.

The authors state in their foreword that the book is intended as a semi-technical or non-technical presentation of the material necessary for an understanding of the effects of ionizing radiation on man. It is intended for the practising physician, the teachers of science in high schools and colleges, medical and dental students, and for others who have not a special training in radiation physics or radiobiology, but for whom it is necessary to have an understanding of the subject of the effects of radiation. There is no complicated physics in the book, and the subject is presented in a series of easily readable chapters which any medical man, specialist, or general practitioner should be able to follow without difficulty.

In the past there has always been the background radiation, in the form of cosmic rays, from the building materials such as stone and brick used in our houses, and there is also radiation in our food to a greater or lesser extent, depending on the nature of the food and on whether the food, vegetables, or animal, have come from a region that has been subjected to 'fall-out' as the result of atom bomb testing.

The development of artificial radioactivity, nuclear power, the ever-increasing use of X-rays for diagnostic and therapeutic purposes, and the introduction of cobalt bombs and linear accelerators, have increased the ionizing radiation to which man is subjected without including the possible dangers of atomic warfare.

The authors state that the number of X-ray machines in use in the world, exclusive of Russia and, one must presume, exclusive of the other Iron Curtain Countries, has increased from approximately 100,000 in 1950 to approximately 225,000 at the present time. It is expected to reach the figure of 300,000 by 1970. This figure may, of course, be reduced if the Governments take an enlightened attitude and impose restriction on the use of X-ray machines by people who have not had some training in radiology.

The authors state that by the end of 1968, there may be 400,000 cobalt units in use. Now, a cobalt unit may of course vary from a relatively small quantity, say 30 - 50 curies, to large quantities such as 3,000 - 5,000 curies. The Oakridge National Laboratory in the United States, which was the first authority to start exporting radioactive materials, and from which South Africa obtained the first radioactive isotopes at the beginning of 1948, exported 25,000 curies in 1952 and 230,000 in 1958. This gives some indication of the increase in the amount of internal ionizing radiation, quite apart from the atomic bomb testing and the subsequent radiation from fall-out material. It must be noted also that a large amount of cobalt for cobalt bomb therapy comes from Canada.

The increase in radiation from these various X-ray sources may sound very terrifying, but it must be realized that this increase in X-radiation spread over whole populations has caused a gonadal dose increase of 19% of the natural background radiation to which we have always been exposed. The authors feel that every physician ought to be aware of the simple facts of the subject. There is a calculated risk in ionizing radiation that we have to balance up against the good, which is being done under certain conditions.

The basic principles concerning radiological hazard are introduced and discussed in the 12 chapters of the book, and while all the chapters are good, some are better than others. Those who have not the time or the inclination to read every chapter, should nevertheless read Chapter 1 (the introduction), Chapter 9 (The clinical picture of acute irradiation injury in man), and Chapter 10 (The diagnosis and therapy of human radiation injury).

The following statement has been stressed over and over again by everybody who has written on this subject of ionizing radiation in the enormous literature which now exists: 'In the use of X-radiation, any potential danger must be weighed against the benefits derived. It is beyond question that X-radiation is indispensable to medical practice and that countless lives are saved and inestimable misery prevented as the result of its use'. A further quotation on the same page, reads: 'Initially, the blunt statement can be made unequivocably that no data are available on human beings at these low-dose levels that allow one to make an estimate with any degree of precision at all'. This being the case, it behoves us to cut out as much unnecessary radiation as possible, but when it has to be used it should be used by those trained to do so.

M.W.

HIGH BLOOD PRESSURE AND PREGNANCY

High Blood Pressure and Pregnancy. By Lance Townsend, M.D., B.S., F.R.C.S. (Edin.), F.R.A.C.S., F.R.C.O.G., D.T.M. & H. Pp. viii+115. 22 figures and 26 tables. R4.00 net. London & New York: Cambridge University Press. 1960.

This book was written by the professor of obstetrics and gynaecology at the University of Melbourne, Australia, and is the thesis submitted by him for the degree of Doctor of Medicine at that university. It is concerned with the effects of pregnancy on the mother and the child in women with high blood pressure.

In an early chapter the author reviews the literature. After dealing with the work of Prof. G. W. Pickering on high blood pressure in general, he reviews the literature appertaining in particular to the problem of the pregnant woman with hypertension.

The actual investigation concerns a series of pregnant women at the Royal Women's Hospital, Melbourne, under the care of the author in 1956. A series of patients treated at the same hospital in the years 1946 - 47 is used as a control. Among his conclusions are the following:

1. Using the method of treatment outlined for the 1956 series, high blood pressure and pregnancy are not dangerous to the mother.
2. The 1956 régime has significantly reduced the perinatal mortality to a figure of 5% or lower.
3. Hypotensive drugs and bed rest are effective in reducing the diastolic blood pressure during pregnancy.
4. It should be the aim of treatment to reduce the diastolic blood pressure below 90 mm.
5. The occurrence of a mid-pregnancy drop in blood pressure is a favourable sign for the foetus and the mother.
6. The initial diastolic blood pressure is a more reliable index to the prognosis for the foetus than is the initial systolic pressure.
7. In patients who commence pregnancy with a high blood pressure, small babies are more commonly found in those with very high diastolic pressures associated with proteinuria at the end of pregnancy.
8. It is the rise or fall in the blood pressure that is important. It is impossible and unnecessary to lay down a normal level of blood pressure for pregnant women.

This book can be recommended for postgraduate reading.

E.M.S.

ANAEMIA

The Haemolytic Anaemias. Congenital and Acquired. Part I—*The Congenital Anaemias*. 2nd edition. By J. V. Dacie, M.D. (Lond.), F.R.C.P. (Lond.). Pp. 399+vii. 118 illustrations. R4.50 net. London: J. & A. Churchill Ltd. 1960.

Such has been the growth of knowledge about haemolytic anaemias in the 6 years since the first edition appeared, that the second edition of Dacie's authoritative work is perforce appearing in two parts. Part I comprises an introductory chapter on the general features and investigation of haemolytic states, and 5 chapters devoted to the congenital haemolytic anaemias and the haemoglobinopathies.

The relationships between these diseases are explained with vivid clarity, and the lucid presentation loses nothing to the encyclopaedic detail which has been marshalled from a huge literature and the author's own considerable researches. The account of hereditary spherocytosis ranges from history through all gradations of clinical and laboratory features, to the latest radio-isotope techniques of diagnosis and a critical consideration of treatment by splenectomy. It is both intensely practical and a fund of information. It would appear to be generally agreed that the anaemic crises in this condition are almost always due to marrow aplasia, the exact mechanism of which is less certain.

A useful chapter deals with certain congenital haemolytic anaemias sometimes confused with hereditary spherocytosis, namely hereditary, non-spherocytic haemolytic anaemia, atypical and unclassified types, 'erythropoietic porphyria' and congenital Heinz-body anaemia.

The description of Mediterranean anaemia and its allied disorders vies with that of the much-lettered and ever-increasing hereditary haemoglobinopathies in its refreshing simplicity. It is interesting to note the evidence that in certain circumstances splenectomy has a definite though restricted place in the treatment of thalassaemia major, as it has also in sickle-cell (Hb-S) disease if the spleen is enlarged in a young child who has to be maintained on transfusions.

Dacie has set out to provide an up-to-date and reasonably complete reference book useful to physicians and pathologists; he has succeeded admirably.

R.S.M.

UROLOGY

Urology in General Practice. By Ian Parton, M.B., Ch.B., B.Sc. (N.Z.), F.R.C.S. (Eng.). Pp. xi+304. 35 illustrations. R4.50 plus R0.20 postage. London: Butterworth and Co. South African Office: Butterworth and Co. (Africa) Ltd., Box 792, Durban. 1960.

This little book on Urology is not for the experts, but as an aid to the general practitioner working on his own. In view of all this it would appear that the scope of its contents is not too over-ambitious. It attempts to survey a wide field covering a great variety of urological conditions and provides advice on their treatment. It also includes a discussion of drugs that will be found useful, while operative procedures are outlined where appropriate. The author has also set himself the task of trying to answer questions, the answers to which are too often taken for granted in larger books. This should enhance the value of his book.

Two features are commendable: The author makes frequent use of short case histories throughout the text; this enables him to emphasize points he tries to bring out in the text. Next, he has seen to it that a useful 'Bibliography and references' appears at the end of the book, again under headings used for chapters. In this section the reader may find sources from which more information may be culled if required.

The book is well written and strongly bound. It can be recommended for use by the newly qualified practitioner.

G.C.A. v.d. W.

YEAR BOOK OF THE EAR, NOSE AND THROAT

The Year Book of the Ear, Nose and Throat, 1959 - 1960. Edited by John R. Lindsay, M.D., with a section on *Maxillofacial Surgery* edited by Dean M. Lierle, M.D. and William C. Huffman, M.D. Pp. 360. 103 figures. $8.00. Chicago: The Year Book Publishers, Inc. 1960.

The Year Book Series is by now sufficiently well known and established to require no introduction to readers. The Editors this year have even dispensed with the customary preface.

The printing and presentation of this year's volume maintains the high standard set in previous years and leaves little room for criticism. Detailed review of the subject matter is difficult, if not impossible, in a volume of this type which aims at presenting abstracts of significant current medical writings. In this regard, in view of the fact that the most exciting and striking developments in Oto-rhino-laryngology today are those taking place in the field of otosclerotic and reconstructive middle ear surgery, I feel that these subjects are very inadequately represented in the abstracts presented in this volume.

All in all, however, it provides a useful bird's eye view of work in progress.

D.V.M.

BRIEWERUBRIEK : CORRESPONDENCE

TRANQULLIZERS

To the Editor: At a refresher course organized by the Transvaal Nursing Education Discussion Group of the South African Nursing Association, which was recently held in Johannesburg, reference was made to the use of tranquillizers and the possible harmful effects when used in excessive doses and when addiction occurs. A resolution was passed that the medical profession be requested to institute an investigation into the effects of tranquillizers.

A male nurse of senior status, whose honesty and integrity cannot

be doubted, and who has much experience in the nursing of psychiatric patients and is thus in a position to report his personal observations of the effects of tranquillizer agents, has registered concern about their harmful effects, especially in the case of 'noludar'* and particularly when taken with alcohol.

The abuse of tranquillizers is causing growing concern among nurses who observe its harmful effects, but it would seem that their concern is not fully shared by all the members of the medical profession.

I use this incident to point out that doctors are very dependent on the trained observation of nurses in their day-to-day medical practice. Nurses are especially trained to observe the effect of drugs on patients and are expected to report any untoward reactions. It is disturbing therefore to hear that this is regarded as 'a meddlesome and unwarranted interference'.**

It would seem to be necessary to realize that the rôle of the nurse has changed. She can no longer be regarded only as the 'handmaiden' of the doctor, but has earned her place as a responsible member of the medical therapeutic team. In treating a patient the doctor gives recognition to the contribution made by the other members of the team; he should likewise be able to draw upon and utilize, for the benefit of the patient, the resources of a large body of workers whose contribution is certainly not intended to be 'meddlesome interference'.

I. I. Marwick

Tara Hospital
P.O. Box 13
Saxonwold
Johannesburg
29 April 1961

[* At its meeting in September 1959, the Federal Council of the Medical Association discussed the question of the inclusion of 'noludar' and 'doriden' in the 6th Schedule of potentially harmful drugs.

** This quotation is taken from a report which appeared in the *Star* of 19 April 1961, and the words are attributed to a 'leading Johannesburg doctor'. — Editor]

COLLEGE OF GENERAL PRACTITIONERS

To the Editor: For your information I have pleasure in advising you of the establishment of an Interim South African Council of the College of General Practitioners, with the following terms of reference:

1. To coordinate the activities of the faculties of the College in South Africa.
2. To supervise and stimulate the work of members and associates of the College in South Africa.
3. To represent the College Council in South Africa.
4. To further the liaison between the College Council and the South African faculties.
5. To comment on applicants for membership of the College from South Africa.
6. To plan financial support for the South African Council of the College.

F. E. Hofmeyr

P.O. Box 643
Cape Town
26 April 1961

REMEDY FOR SANDWORM

To the Editor: With reference to the letter on the above subject, which was published in the *Journal* for 15 April,[1] I previously reported the successful results of treatment with stovarsol.[2]

Since that date I have treated about 50 - 60 cases *per annum* with stovarsol, and only twice have I reverted to the use of an ethyl chloride spray—in both cases because of impatience on the part of the parents. It is essential that all signs of sepsis should be eliminated before the administration of the stovarsol. Occasionally, after the activity of the 'worm' has ceased, the track develops a bright red colour and becomes very itchy. This is relieved by any antihistaminic and is, I think, due to an allergic reaction to toxic products.

The treatment is as follows:

(*a*) For a child over six or an adult, 4 gr. *t.d.s.* for 4 days (12 tablets).

(*b*) For a child under 6, 2 gr. *t.d.s.* for 6 days.

Occasionally, in younger children, diarrhoea is a complication of the treatment, but this can be ignored unless it is severe and occurs early in the treatment; if this happens, the rate of administration is reduced. On two occasions, both in adults, I have seen generalized erythematous rashes which subsided spontaneously at the end of treatment. Incidentally, I have also found stovarsol, administered as above, very useful in scabies.

Thirty years ago, the internal administration of arsenic accompanied topical treatment for many dermatological conditions, but this seems to have been largely forgotten. I still find it very useful.

Philip H. Dalgleish

Hill Crest
Natal
22 April 1961

1. Correspondence (1961): S. Afr. Med. J., 35, 320.
2. *Idem* (1950): *Ibid.*, 24, 991.

ADMISSION OF MEDICAL AID PATIENTS TO TEACHING HOSPITALS

To the Editor: The following is the relevant portion of a letter which I received from the Director of Hospital Services, Cape Town:

'It is not correct that this Department has categorically laid down that membership of a medical aid society should not debar any patient from admission to any provincial hospital. The Department merely pointed out to the closed hospitals that there might be circumstances which justified the admission of a medical aid society member to a closed hospital. The hospitals were, therefore, requested not to exclude such patients *automatically*. As in the case of all other patients, a certificate of inability to pay for private medical treatment is demanded and it is presumed that, before issuing such a certificate, medical practitioners will satisfy themselves that the patient's society benefits have been exhausted or that he needs specialized treatment not available elsewhere.'

A. R. R. Mears
Hon. Secretary

Cape Western Branch (M.A.S.A.)
P.O. Box 643
Cape Town
25 April 1961

NAUDE APPEAL FUND

To the Editor: With regard to the Naude Appeal Fund, we should appreciate it if you would kindly inform members of the Association, through the *Journal*, that it has been decided by the Transkei Branch to close the Fund.

We should like to thank you and all members of the Medical Association of South Africa for your cooperation in connection with this Appeal Fund.

J. H. Hofmeyr
Hon. Secretary

Transkei Branch (M.A.S.A.)
Umtata
19 April 1961

THE LOUIS MIRVISH MEMORIAL MEDICAL FOUNDATION

To the Editor: The response to the appeal for funds to the above Foundation has been most gratifying. There are still many members of the Association, however, who have not responded, and I would appreciate it if I could, through the medium of your columns, ask them to send their donations to Barclay's Bank D.C.O., Heerengracht Branch, P.O. Box 4671, Cape Town, as soon as possible, so that the matter can be finalized.

R. L. Tobias

Medical Centre
Cape Town
26 April 1961